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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
43RD YEAR—ESTABLISHED 1956

SPECIAL ISSUE

SLEEP-DISORDERED BREATHING: PART I

Final Deadline for 1998 OPEN FORUM
Abstracts: May 4, 1998
Now Available—Electronic Submission

SEE PAGE 333

CONFERENCE PROCEEDINGS

Foreword:
Susceptibility, Strategies of Care, & Schooling in Sleep-Disordered Breathing

Pathogenesis of Obstructive Sleep Apnea

Consequences of Sleep-Disordered Breathing

Clinical Diagnosis of Sleep Apnea

Sleep Testing:
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110	111	112	113	114	115	116	117	118	119	120	121	122	123	124
125	126	127	128	129	130	131	132	133	134	135	136	137	138	139
140	141	142	143	144	145	146	147	148	149	150	151	152	153	154
155	156	157	158	159	160	161	162	163	164	165	166	167	168	169
170	171	172	173	174	175	176	177	178	179	180	181	182	183	184
185	186	187	188	189	190	191	192	193	194	195	196	197	198	199

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110	111	112	113	114	115	116	117	118	119	120	121	122	123	124
125	126	127	128	129	130	131	132	133	134	135	136	137	138	139
140	141	142	143	144	145	146	147	148	149	150	151	152	153	154
155	156	157	158	159	160	161	162	163	164	165	166	167	168	169
170	171	172	173	174	175	176	177	178	179	180	181	182	183	184
185	186	187	188	189	190	191	192	193	194	195	196	197	198	199

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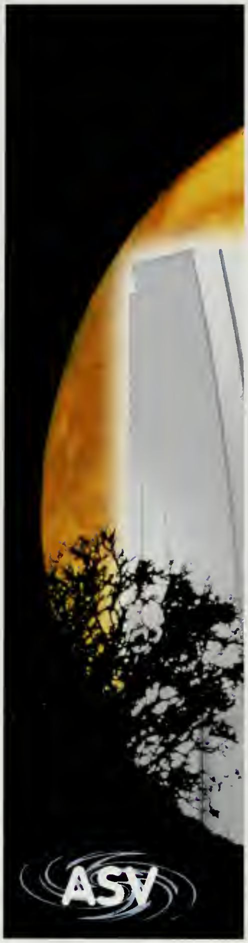

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CONTENTS

APRIL 1998 / VOLUME 43 / NUMBER 4

SPECIAL ISSUE

SLEEP-DISORDERED BREATHING—PART I

The proceedings of a conference
held in Cancún, Mexico, October 17-19, 1997

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Patrick J Strollo Jr MD and James K Stoller MD

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CONFERENCE PROCEEDINGS

Sleep-Disordered Breathing:
The 1997 Respiratory Care Journal Conference **262**

Foreword—Susceptibility, Strategies of Care, and Schooling in Sleep-Disordered Breathing: The 1997 RESPIRATORY CARE Journal Conference
by James K Stoller and Kingman P Strohl—Cleveland, Ohio, and Patrick J Strollo Jr—Pittsburgh, Pennsylvania **264**

Pathogenesis of Obstructive Sleep Apnea
by Alejandro D Chediak—Miami Beach, Florida **265**

Consequences of Sleep-Disordered Breathing
by Kingman P Strohl—Cleveland, Ohio **277**

Clinical Diagnosis of Sleep Apnea
by Barbara A Phillips—Lexington, Kentucky **288**

Sleep-Testing: Center- and Home-Based
by Pamela Minkley—Lansing, Michigan **296**

Positive Pressure Therapy: Theory and Application
by Charles W Atwood Jr—Pittsburgh, Pennsylvania **307**

Positive Pressure Therapy: Compliance and Partnering with Home Care
by Karen A Studnicki—Pittsburgh, Pennsylvania **318**

ALSO IN THIS ISSUE

329 AARC Membership Application

246 Abstracts from Other Journals

344 Advertisers Index & Help Lines

344 Author Index

339 Calendar of Events

333 Call for OPEN FORUM Abstracts

326 Correction *Respir Care 1998; 43(2):107-113*

335 Manuscript Preparation Guide

331 MedWatch

340 New Products & Services

342 Notices

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Abstracts

Summaries of Pertinent Articles in Other Journals

Editorials, Commentaries, and Reviews To Note

Asthma Disease Management (Editorial)—Homer CJ. N Engl J Med 1997;337(20):1461-1463.

Challenges in Pulmonary Medicine: Beating Bronchitis and Surviving Septic Shock (Supplement)—Niederman M, Chernow B, Dinarello C, editors. Chest 1997;112(6):3015-3445.

Obstructive Sleep Apnoea: A Progressive Disorder? (Editorial)—Calverley PM. Thorax 1997 Oct;52(10):843-844.

Principles of Judicious Use of Antimicrobial Agents for Pediatric Upper Respiratory Tract Infections (Supplement)—Dowell SF, editor. Pediatrics 1998;101(1):163-184.

The Significance of Sleep-Disordered Breathing and Obstructive Sleep Apnea in the Elderly (Editorial)—Collop NA. Chest 1997;112(4):867-868.

Treating Tobacco Addiction—Nicotine or No Nicotine? (Editorial)—Benowitz NL. N Engl J Med 1997;337(17):1230-1231.

Vancomycin-Resistant *Staphylococcus aureus:* Apocalypse Now? (Comment)—Tabaqchali S. Lancet 1997;350(9092):1644-1645.

Effect of Inhaled Formoterol and Budesonide on Exacerbations of Asthma—Formoterol and Corticosteroids Establishing Therapy (FACET) International Study Group. Pauwels RA, Lofdahl CG, Postma DS, Tattersfield AE, O'Byrne P, Barnes PJ, Ullman A. N Engl J Med 1997;337 (20):1405.

BACKGROUND: The role of long-acting, inhaled β-2 agonists in treating asthma is uncertain. In a double-blind study, we evaluated the effects of adding inhaled formoterol to both lower and higher doses of the inhaled glucocorticoid budesonide. METHODS: After a 4-week run-in period of treatment with budesonide (800 μg twice daily), 852 patients being treated with glucocorticoids were randomly assigned to 1 of 4 treatments given twice daily by means of a dry-powder inhaler (Turbuhaler): 100 μg of budesonide plus placebo, 100 μg of budesonide plus 12 μg of formoterol, 400 μg of budesonide plus placebo, or 400 μg of budesonide plus 12 μg of formoterol. Terbutaline was permitted as needed. Treatment continued for 1 year; we compared the frequency of exacerbations of asthma, symptoms, and lung function in the 4 groups. A severe exacerbation was defined by the need for oral glucocorticoids or a decrease in the peak flow to more than 30% below the baseline value on 2 consecutive days. RESULTS: The rates of severe and mild exacerbations were reduced by 26% and 40%, respectively, when formoterol was added to the lower dose of budesonide. The higher dose of budesonide alone reduced the rates of severe and mild exacerbations by 49% and 37%, respectively. Patients treated with formoterol and the higher dose of budesonide had the greatest reductions—63% and 62%, respectively. Symptoms of asthma and lung function improved with both formoterol and the higher dose of budesonide, but the improvements with formoterol were greater. CONCLUSIONS: In patients who have persistent symptoms of asthma despite treatment with inhaled glucocorticoids, the addition of formoterol to budesonide therapy or the use of a higher dose of budesonide may be beneficial. The addition of formoterol to budesonide therapy improves symptoms and lung function without lessening the control of asthma.

Lung Structure and Ventilation in Theropod Dinosaurs and Early Birds—Ruben JA, Jones TD, Geist NR, Hillenius WJ. Science 1997;278 (5341):1267.

Reptiles and birds possess septate lungs rather than the alveolar-style lungs of mammals. The morphology of the unmodified, bellowslike septate lung restricts the maximum rates of respiratory gas exchange. Among taxa possessing septate lungs, only the modified avian flow-through lung is capable of the oxygen-carbon dioxide exchange rates that are typical of active endotherms. Paleontological and neontological evidence indicates that theropod dinosaurs possessed unmodified, bellows-like septate lungs that were ventilated with a crocodile-like hepatic-piston diaphragm. The earliest birds (Archaeopteryx and enantiornithines) also possessed unmodified septate lungs but lacked a hepatic-piston diaphragm mechanism. These data are consistent with an ectothermic status for theropod dinosaurs and early birds.

Health Effects of Passive Smoking. I. Parental Smoking and Lower Respiratory Illness in Infancy and Early Childhood—Strachan DP, Cook DG. Thorax 1997;52(10):905.

BACKGROUND: A systematic quantitative review was conducted of evidence relating parental smoking to acute lower respiratory illness in the first 3 years of life. METHODS: Fifty relevant publications were identified after consideration of 692 articles selected by electronic search of the Embase and MEDLINE databases using keywords relevant to passive smoking in children. The search, completed in April 1997, identified 24 studies ascertaining illnesses in a community setting, including 5 surveys of schoolchildren with retrospective ascertainment of early chest illness, and 17 studies of admissions to hospital for lower respiratory illness in early life. Thirty-eight studies were included in a quantitative overview using random effects modelling to derive pooled odds ratios. RESULTS: The results of community and hospital studies are broadly consistent, with only 1 publication reporting a reduced risk among children of smokers. The pooled odds ratios were 1.57 (95% confidence interval [CI] 1.42 to 1.74) for smoking by either parent and 1.72 (95% CI 1.55

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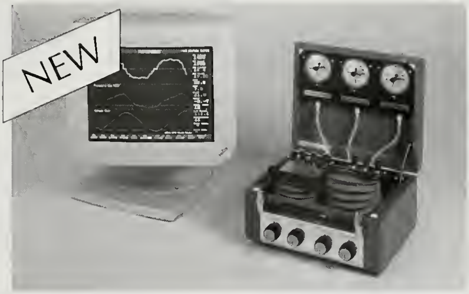

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to 1.91) for maternal smoking. There is a significantly increased risk of early chest illness associated with smoking by other household members in families where the mother does not smoke (1.29, 95% CI 1.16 to 1.44). The associations with parental smoking are robust to adjustment for confounding factors, and show evidence of a dose-response relationship in most studies in which this has been investigated. CONCLUSIONS: The relationship between parental smoking and acute lower respiratory illness in infancy is very likely to be causal. Although it is impossible to distinguish the independent contributions of prenatal and postnatal maternal smoking, the increased risk associated with smoking by other household members suggests that exposure to environmental tobacco smoke after birth is a cause of acute chest illness in young children.

Validation of Preferences for Life-Sustaining Treatment: Implications For Advance Care Planning—Patrick DL, Pearlman RA, Starks HE, Cain KC, Cole WG, Uhlmann RF. Ann Intern Med 1997;127(7):509.

BACKGROUND: Treatment preferences established before life-threatening illness occurs may differ from actual decisions because of changes in preferences or poor understanding of the link between prospective preferences and outcomes. OBJECTIVES: To evaluate the validity of prospective treatment preferences by examining their concordance with ratings of health states. DESIGN: Survey of 7 cohorts of persons with diverse health status. Home- and hospital-based interviews were conducted at baseline and at 6, 18, and 30 months. SETTING: The greater Seattle area. PARTICIPANTS: Younger and older well adults; persons with chronic conditions, terminal cancer, or acquired immunodeficiency syndrome (AIDS); stroke survivors; and nursing home residents. MEASUREMENTS: Concordance between 6 treatment preferences and 5 health state ratings (on a 7-point scale) was assessed by using logistic regression to measure the increase in odds of treatment refusal for each 1-point change in health state rating. Preferences were considered concordant if treatments were refused in health states rated as worse than death and were accepted in health states rated as better than death. Reasons for discordance were elicited at the final interview. RESULTS: The probability of refusal of prospective treatment was strongly related to health state ratings. Odds ratios ranged from 1.7 to 1.9 ($p < 0.001$) for every treatment. When patients were shown their discordant preferences, they had a coherent explanation or changed their health state rating or treatment preference to make the two concordant. CONCLUSIONS: Prospective life-sustaining treatment preferences show high convergent validity. For most persons, treatment preferences are grounded in a consistent belief system. Concordance and discordance between treatment preferences and health state ratings offer clinicians the opportunity to explore patients' values and reasoning.

Prevention of Pulmonary Morbidity for Patients with Duchenne Muscular Dystrophy—Bach JR, Ishikawa Y, Kim H. Chest 1997;112(4):1024.

OBJECTIVE: To evaluate the effects of a new respiratory management protocol on respiratory morbidity and hospitalization rates for patients with Duchenne muscular dystrophy (DMD). DESIGN: A retrospective cohort study. METHODS: Using a protocol in which oxyhemoglobin desaturation was prevented or reversed by the use of noninvasive intermittent positive pressure ventilation (IPPV) and assisted coughing as needed, the hospitalization rates and days for 24 protocol DMD ventilator users were compared with those of 22 nonprotocol DMD tracheostomy IPPV users. RESULTS: The 22 conventionally managed patients were hospitalized a mean of 72.2 ± 112 days when undergoing tracheostomy. This included a 16.1

± 5.4-day period of translaryngeal intubation. The 24 protocol patients were hospitalized a mean of 6.0 ± 2.4 days ($p < 0.005$) when beginning ventilator use. Over their next 126.2 patient-years of ventilator use, the 24 protocol patients had significantly lower rates of hospitalization ($p < 0.008$) and hospitalization days ($p < 0.005$) than had the tracheostomy IPPV users over a 167.2 patient-year period. This is true although 14 of the 24 protocol patients went on to require 24-hour noninvasive IPPV for 4.5 ± 3.6 years. Five of the 14 have yet to be hospitalized. CONCLUSION: The use of inspiratory and expiratory aids can prolong survival while significantly decreasing the pulmonary morbidity and hospitalization rates associated with conventional resort to tracheostomy IPPV.

Women and Lung Cancer: Waiting to Exhale—Baldini EH, Strauss GM. Chest 1997;112(4 Suppl):229S.

Lung cancer is now the leading cause of cancer deaths among women. In the United States, 64,300 women are expected to die of lung cancer in 1996. Smoking is responsible for about 80% of lung cancer cases. Unfortunately, the prevalence of smoking among women remains unacceptably high at about 22% and is expected to surpass the rate in men by the year 2000. Smoking rates are highest among young girls and the less educated. Whether lung cancer represents a different disease in women than in men is unclear. Data are conflicting regarding the magnitude of the relative risk of developing lung cancer due to smoking between the genders. There appears to be a difference in the relative distribution of lung cancer histologic features between men and women that is not explained entirely by differences in smoking patterns. Women who smoke appear to be at higher risk of developing small cell lung cancer than squamous cell lung cancer, whereas men who smoke have a similar risk for the 2 histologic conditions. Furthermore, women smokers are more likely to develop adenocarcinoma of the lung, and estrogens may play a causative role in this phenomenon. Data are unclear regarding whether the outcome of lung cancer treatment differs between genders. Solutions to the lung cancer epidemic among U.S. women include (1) prevention of the disease by reducing smoking rates, (2) improving early detection methods, and (3) exploring new therapeutic strategies.

Sputum and Pulmonary Function in Asthma—Connolly CK, Murthy NK, Alcock SM, Prescott RJ. Chest 1997;112(4):994.

OBJECTIVE: To assess the relevance of sputum production to pulmonary function, in particular, persistent obstruction in patients with a primary clinical diagnosis of asthma. DESIGN: Cross-sectional study of all patients currently followed up in secondary care in a defined locality. SETTING: National Health Service and private clinics in northeast England. PATIENTS: All attenders, aged 18 years or older, with asthma, confirmed by reversibility of peak expiratory flow (PEF) by ≥ 15% and to ≥ 200 L/min. INTERVENTIONS: Pro forma history. Pulmonary function at attendance. Assessment of best function according to protocol. Measurement of actual FEV_1, FVC, and PEF at attendance. MEASUREMENTS & RESULTS: We studied 772 subjects; 387 (50%) were male; mean age was 55 years; atopic, 51%; current smokers, 11.5%; ex-smokers, 36%; and never smokers, 52.5%. Best pulmonary function was lower in chronic sputum producers (PEF, 83.2 vs 95.8; FVC, 67.9 vs 81.7% predicted). Chronic sputum production and its negative relationship with best function was strongly associated with smoking. There was little relationship between chronic sputum and persistent obstruction in nonsmokers. There were no univariate associations between sputum during attacks, or its color, and pulmonary function, but after allowing for demographic factors, including smoking, green sputum was associated with persistent obstruction. There was little relationship between sputum and actual/best function at attendance. CONCLUSIONS: Chronic sputum production is associated with persistent obstruction principally in those who have smoked, suggesting that the association reflects smoking rather than asthma. There is no interaction with atopy. After allowance for cigarette smoking, there is an association between green sputum production during exacerbations and persistent obstruction. Green sputum during relapse in asthma may indicate inflammation that is relevant to prognosis.

Natural Evolution of Moderate Sleep Apnoea Syndrome: Significant Progression over a Mean of 17 Months—Pendlebury ST, Pepin JL, Veale D, Levy P. Thorax 1997;52(10):872.

BACKGROUND: Obstructive sleep apnoea (OSA) is associated with increased morbidity and mortality. It has remained unclear whether or not it is progressive. The evolution of OSA was examined in a retrospective case note study of 55 unselected patients of mean (standard deviation) age 55.8 (10) years with mild to moderate disease untreated by interventional methods such as continuous positive airway pressure (CPAP) or surgery. Correlations between clinical and functional variables, upper airway anatomy, and change in disease severity were also investigated. METHODS: Patients underwent full polysomnography on two occasions (T_0 and T_x) at a mean interval of 77 (50) weeks (range 17-229). In addition, upper airway imaging with computed tomographic scanning or cephalometry had been performed in 43 patients at T_0. Morbidity before, during, and after the study period was assessed by questionnaire, as was smoking history and alcohol and sedative intake. RESULTS: The apnoea hypopnoea index (AHI) for the group as a whole increased from 21.8 (11.5) to 33.4 (21.3) ($p = 0.0001$). Using a 25% change in AHI to divide patients into worsened, stable, and improved groups showed that, although most of the patients deteriorated, 25 patients improved or remained stable. The change in AHI was not correlated with body mass index which remained stable at 29.7 (5.4) kg/m^2 versus 29.7 (5.6) kg/m^2. There was a trend for apnoea duration to increase. No patient reported increased alcohol consumption and only 1 patient reported increased use of sedatives between T_0 and T_x. No correlation was found between change in AHI and age, time between recordings, anatomical measurements of the upper airway, respiratory function, oximetry, or arterial blood gas tensions. Total cardiovascular and cerebrovascular morbidity was high: hypertension (26 patients, 46%), cardiac arrhythmia (17 patients, 33%), angina (12 patients, 23%), myocardial infarction (10 patients, 19%), and stroke (10 patients, 19%). Twenty-nine patients (52%) were prescribed CPAP after T_x, two of whom went on to have maxillofacial surgery. These 29 treated patients had significantly higher values of AHI at T_0 and T_x and greater change in AHI than the untreated patients. CONCLUSIONS: This study shows that mild to moderate OSA has a tendency to worsen in the absence of significant weight gain and that upper airway anatomy and clinical variables do not appear to be useful in predicting progression. It follows that mild to moderate OSA justifies systematic follow up. Deterioration in AHI over a mean of 17 months led to interventional treatment in over 50% of patients in the study.

Nonprescription Bronchodilator Medication Use in Asthma—Kuschner WG, Hankinson TC, Wong HH, Blanc PD. Chest 1997;112(4):987.

OBJECTIVE: Many persons with asthma self-medicate with widely available and potentially hazardous nonprescription medicines. This study assessed the demographic and clinical covariates of self-treatment with over-the-counter asthma medications (OTCs). DESIGN & SETTING: We conducted an analytical investigation using questionnaires and measures of lung function, comparing OTC and prescription medication users. We recruited adults with asthma by public advertisement. SUBJECTS: We studied 22 exclusive prescription asthma medication users, 15 exclusive OTC users, and 13 other subjects who combined prescription medication use with self-treatment with asthma OTCs. All but 1 OTC user self-medicated with a nonselective, sympathomimetic metered dose inhaler. RESULTS: Taking income, access to care, and self-assessed disease severity into account, male gender was strongly associated with exclusive OTC use alone (odds ratio [OR] = 8.9, 95% confidence interval [CI] = 1.3 to 61) and mixed OTC-prescription medication use (OR = 9.7, 95% CI = 1.1 to 83). The covariates of income, access to care, and self-assessed disease severity provided significant additional explanatory power to the model of exclusive OTC use (model χ^2 difference 11.3, 5 df, $p < 0.05$). Pulmonary function was similar among OTC and pre-

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REFERENCES: 1. Data on file, HealthScan Products Inc. Results of aerosol testing with albuterol, beclomethasone, and cromolyn sodium. 2. Data on file, HealthScan Products Inc. Results of gamma-scintigraphy aerosol deposition studies. 3. Gibson PG, Wlodarczyk JH, Borgas T: Drug delivery in asthma: A comparison of spacers with a jet nebuliser. *Aust NZ J Med* 25:324-329, 1995.

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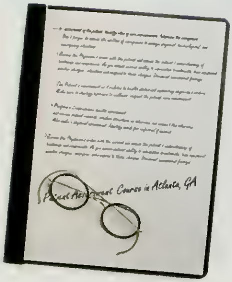

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AARC Assessment Course/Examination Detailed Content Outline

1. Obligations of the Respiratory Care Practitioner in the Practice of Patient Assessment

A. Appreciate the cost of care
B. Understand the scope and limitations of the practitioners role in patient assessment
C. Understand the principle of patient self-determination
D. Appreciate the psychological, social, and physical characteristics of wellness/illness
E. Recognize the patient's right to confidentiality
F. List the principles of informed consent

▶ "Assessment of the patient isn't purely clinical anymore — helped to start the 'Thinking outside the box concept,' that is necessary for continued survival of RT as a profession."

2. Perform a Comprehensive Health Assessment

A. Review patient records
B. Structure an interview
C. Conduct an interview
D. Conduct a physical assessment
E. Identify needs for referral
F. Identify patients with potential for high-risk medical complications
G. Accurately document assessment findings
H. Present findings to a physician

▶ "I truly believe that assessment (total — not just physical) is what will keep our profession a vital portion of the health care team."

3. Assess the Caregivers

A. Identify sites of care and caregivers
B. Interview the caregivers
C. Assess the abilities of caregivers to manage physical, technological, and emergency situations
D. Assess home health resources

4. Assess the Patient's Understanding and Compliance with Physician's Orders and Care Plan

A. Review the physician's order with the patient
B. Assess the patient's understanding of technique and components

▶ "I plan on sending other staff who will be involved in asthma case management to the April '98 course in Dallas"

John Kimble, RT
Director, Respiratory Care Services and Sleep Disorders Laboratory, Scott & White Memorial Hospital, Temple, TX

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▶ "I traveled ten hours to get here and feel it was well worth the trip. Your program is great."
Penny Gagne Plouff, RRT
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C. Assess patient ability to administer treatments, use equipment, monitor changes, interpret and respond to those changes
D. Document assessment findings

5. Assess the Patient's Environment as It Relates to Health Status and Supporting Physician's Orders

A. Identify barriers to wellness

▶ "The course was relevant to the changes in health care and my practice. Well worth my time."

B. Inspect the patient care environment
C. Determine the resources available to the patient and family
D. Document assessment findings

▶ "Bravo for a job well done. I plan to share a lot of this information with staff."

6. Assess Continued Appropriateness of Physician Orders and Care Plan

A. Review physician orders
B. Evaluate the patient's response to physician orders
C. Evaluate the appropriateness of the orders and the care plan
D. Recommend modification of the treatment plan
E. Document assessment findings

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scription medication users. However, prescription medication users' self-assessed asthma severity (mild compared to more severe) was associated with postbronchodilator reversibility of FEV_1 obstruction (6% vs 18% reversibility, $p < 0.05$) while exclusive OTC users' self-assessed severity showed the reverse pattern (19% vs 8%, $p = 0.2$). CONCLUSION: Asthma education programs attempting to discourage unregulated bronchodilator use should give consideration to this profile of the "asthmatic-at-risk."

Transmission of *Mycobacterium tuberculosis* by a Fiberoptic Bronchoscope. Identification by DNA Fingerprinting—Michele TM, Cronin WA, Graham NM, Dwyer DM, Pope DS, Harrington S, et al. JAMA 1997;278(13):1093.

CONTEXT: An ongoing restriction fragment length polymorphism (RFLP) study of *Mycobacterium tuberculosis* isolates from tuberculosis (TB) cases revealed an identical 10-banded IS6110 RFLP pattern unique to 2 patients diagnosed as having TB 6 months apart. Their only identifiable link was care at the same hospital. OBJECTIVE: To determine if nosocomial transmission had occurred. DESIGN: Traditional and molecular epidemiologic investigation. MEASUREMENTS: We reviewed medical charts and bronchoscopic records, examined hospital locations visited by both patients, evaluated hospital ventilation systems, and observed cleaning and disinfection of bronchoscopes. RESULTS: A patient with cough, hoarseness, and fever underwent bronchoscopy and was diagnosed as having TB. A second patient with a mediastinal mass underwent bronchoscopy 2 days later and was diagnosed as having small cell carcinoma. Following 6 months of chemotherapy and radiation therapy, the second patient developed fever and an infiltrate of the right upper lobe of the lung. Bronchoscopic washings revealed acid-fast bacilli and were culture positive for *M. tuberculosis*. Both patients had undergone bronchoscopy with the same instrument in the same operating room with no intervening bronchoscopies. Bronchoscope cleaning and disinfection procedures were inconsistent with national guidelines. CONCLUSIONS: A contaminated bronchoscope was the most likely source of *M. tuberculosis* transmission between these 2 patients. The RFLP analysis of *M. tuberculosis* isolates was responsible for detecting this nosocomial source of transmission and led to the implementation of public health measures to prevent further spread of infection and disease. This study emphasizes the need for continued vigilance in endoscope cleaning techniques.

Serial Measurements of the Rapid-Shallow-Breathing Index as a Predictor of Weaning Outcome in Elderly Medical Patients—Krieger BP, Isber J, Breitenbucher A, Throop G, Ershowsky P. Chest 1997;112(4):1029.

OBJECTIVES: To determine the usefulness of serial measurements of the rapid-shallow-breathing index (f/V_T) as a predictor for successfully weaning elderly medical patients from mechanical ventilator support using a threshold value (≤ 130) derived specifically for this population. DESIGN: Prospective observational study using parameters suggested from retrospective analysis.

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SETTING: Medical ICUs of a university-affiliated private teaching hospital. PATIENTS: Using data obtained from a retrospective analysis of 10 medical patients ≥ 70 years old who had failed weaning, 49 additional medical patients older than 70 years were studied prospectively. INTERVENTIONS: Standard weaning parameters were determined using a hand-held spirometer. Respiratory rate (f, breaths/min) and tidal volume (V_T, L) were measured at the beginning of a spontaneous breathing trial and hourly thereafter for up to 5 hours using the same hand-held spirometer. MEASUREMENTS & RESULTS: Retrospective analysis showed that the published threshold value for f/V_T (≤ 105) had poor predictability for weaning success when measured at the beginning of the weaning trial. In the 9 of 10 patients who failed to wean in the retrospective review, the f/V_T increased to > 130 as the trial progressed over 2 to 3 hours. Using an $f/V_T \leq 130$ as the threshold value for prospectively predicting successful weaning, the diagnostic accuracy, sensitivity, specificity, positive predictive value, and negative predictive value increased from 84%, 92%, 57%, 87%, and 67%, respectively, when measured at the beginning of the weaning trial to 92%, 93%, 89%, 97%, and 80%, respectively, when measured 3 hours later. The area under the receiver operating characteristic curve for f/V_T also improved from 0.81 to 0.93. CONCLUSIONS: Serial measurements of the rapid-shallow-breathing index in medical elderly patients during a period of spontaneous breathing can accurately predict the ability to be successfully weaned from mechanical ventilator support.

Total Daily Energy Expenditure Relative to Resting Energy Expenditure in Clinically Stable Patients with Chronic Obstructive Pulmonary Disease (COPD)—Baarends EM, Schols AM, Westerterp KR, Wouters EF. Thorax 1997; 52(9):780.

BACKGROUND: An elevated resting energy expenditure (REE) commonly occurs in patients with chronic obstructive pulmonary disease (COPD). The purpose of this study was to investigate the effect of an increased REE on total daily energy expenditure (TDE) in 20 patients with COPD (19 men) with mean (standard deviation) forced expiratory volume in one second of 37 (14)% predicted. METHODS: TDE was measured over a 2 week interval using doubly labelled water. Fat-free mass (FFM) was calculated from total body water assessed by deuterium dilution. REE was measured by indirect calorimetry using a ventilated hood system. RESULTS: The patients (10 men) with a significantly higher REE than those with a normal REE (median difference 205 kcal/24 h, $p < 0.05$) had a comparable TDE (hypermetabolic at rest: median 2593; range 2127-3083 kcal/24 h, normometabolic at rest: median 2629; range 2032-3179 kcal/24 h). There was no difference in mean daily heart rate (HR) between the groups (hypermetabolic at rest: median 92 (range 82-98), normometabolic at rest: median 98 (range 75-116 beats/min) or in the variation in the heart rate during the day. By means of multiple regression analysis it was shown that REE did not correlate significantly with TDE when FFM was taken into account. CONCLUSIONS: This study shows that there is no significant difference in free living TDE between clinically stable patients with COPD with a normal REE and those with an increased REE. The variation in TDE in patients with COPD appears to reflect differences in energy expenditure for activities, but not differences in REE.

The Effect of Medicare's Payment System for Rehabilitation Hospitals on Length of Stay, Charges, and Total Payments—Chan L, Koepsell TD, Deyo RA, Esselman PC, Haselkorn JK, Lowery JK, Stolov WC. N Engl J Med 1997;337 (14):978.

BACKGROUND: Medicare's system for the payment of rehabilitation hospitals is based on limits derived from a hospital's average allowable charges per patient discharged during a base year. Thereafter, payments are capped but hospitals receive incentive payments if charges per patient are reduced in succeeding years. We hypothesized that per-patient charges would increase during the base year and then decrease in subsequent years. Hospitals would thus have higher reimbursement limits and receive incentive payments for reducing their charges. METHODS: We analyzed Medicare claims data for 190,921 discharges from 69 rehabilitation hospitals from 1987 through 1994. We compared total charges, length of stay (LOS), and interim payments before, during, and after each hospital's base year. RESULTS: After we controlled for inflation and temporal and seasonal trends, mean charges per patient discharged increased from $25,131 for patients discharged before the base year to $32,167 for patients discharged in the base year (a 28% increase, $p < 0.001$) and the mean LOS increased from 22.1 to 26.7 days (a 21% increase, $p < 0.001$). After the base year, mean charges decreased to $29,307 (a 9% decrease) and the mean LOS decreased to 24.0 days (a 10% decrease) ($p < 0.001$ for both comparisons). Analysis of data on patients according to diagnosis—for example, spinal cord injury, brain injury, stroke, amputations and deformities, hip fracture, and arthritis and joint disorders—showed similar findings for each, with increases in charges and LOS in the base year, followed by smaller reductions thereafter. For-profit hospitals had greater increases than nonprofit hospitals in their per-patient charges (mean increase, $7,434 vs. $2,929; $p < 0.001$) and LOS (mean increase, 4.6 vs. 2.3 days, $p < 0.001$) during the base year. CONCLUSIONS: Although Medicare's reimbursement system for rehabilitation hospitals put an upper limit on total payments, its design was associated with substantial extra costs, including significantly increased payments to hospitals and doctors and increased numbers of hospital days for the average patient.

Urban Air Pollution and Emergency Admissions for Asthma in Four European Cities: The APHEA Project—Sunyer J, Spix C, Quenel P, Ponce-de-Leon A, Ponka A, Barumandzadeh T, et al. Thorax 1997;52(9):760.

BACKGROUND: A study was undertaken to assess the combined association between urban air pollution and emergency admissions for asthma during the years 1986-92 in Barcelona, Helsinki, Paris and London. METHODS: Daily counts were made of asthma admissions and visits to the emergency room in adults (age range 15-64 years) and children (< 15 years). Covariates were short-term fluctuations in temperature and humidity, viral epidemics, day of the week effects, and seasonal and secular trends. Estimates from all the cities were obtained for the entire period and separately by warm or cold seasons using Poisson time-series regression models. Combined associations were estimated using meta-analysis techniques. RESULTS: Daily admissions for asthma in adults increased significantly with increasing ambient levels of nitrogen dioxide (NO$_2$) (relative risk [RR] per 50 μg/m^3 increase 1.029, 95% [confidence interval (CI)] 1.003 to 1.055) and nonsignificantly with particles measured as black smoke (RR 1.021, 95% CI 0.985 to 1.059). The association between asthma admissions and ozone (O$_3$) was heterogeneous among cities. In children, daily admissions increased significantly with sulphur dioxide (SO$_2$) (RR 1.075, 95% CI 1.026 to 1.126) and nonsignificantly with black smoke (RR 1.030, 95% CI 0.979 to 1.084) and NO$_2$, though the latter only in cold seasons (RR 1.080, 95% CI 1.025 to 1.140). No association was observed for O$_3$. The associations between asthma admissions and NO$_2$ in adults and SO$_2$ in children were independent of black smoke. CONCLUSIONS: The evidence of an association between air pollution at current urban levels and emergency room visits for asthma has been extended to Europe. In addition to particles, NO$_2$ and SO$_2$—by themselves or as a constituent of a pollution mixture—may be important in asthma exacerbations in European cities.

Pressure-Controlled Ventilation in Acute Respiratory Distress Syndrome (ARDS): A Practical Approach—Marik PE, Krikorian J. Chest 1997;112(4):1102.

Patients with acute respiratory distress syndrome (ARDS) typically have functionally small lungs. A growing body of clinical and experimental evidence has demonstrated that mechanical ventilation that results in high transpulmonary pressure gradients and overdistention of lung units will potentiate the acute lung injury in patients with ARDS. A relative form of "lung rest" using low tidal volume mechanical ventilation that prevents alveolar overdistention has therefore been advocated. This may be achieved with low-volume, volume-cycled ventilation with a decelerating inspiratory flow or pressure-controlled ventilation (PCV). The goal of this article is to provide a simple and practical approach to the management of PCV in patients with ARDS. Implicit in our approach is the use of a ventilator with PCV software and waveform capabilities.

Salbutamol Delivery from a Hydrofluoroalkane Pressurized Metered-Dose Inhaler in Pediatric Ventilator Circuits: An In Vitro Study—Wildhaber JH, Hayden MJ, Dore ND, Devadason SG, LeSouef PN. Chest 1998;113(1):186.

OBJECTIVES: The aim of our study was to determine the in vitro delivery of salbutamol from a pressurized metered dose inhaler (pMDI) containing hydrofluoroalkane (HFA) propellant through various delivery devices to 4 models of a pediatric lung. DESIGN: To determine the effect of electrostatic charge, delivery of salbutamol was initially assessed with a multistage liquid impinger (MSLI) through an in-line nonchamber device (Baxter MDI Adapter) and a small (Aerochamber MV) and a large (Nebuhaler) in-line chamber device. Following this, the delivery was assessed to 4 lung models appropriate for a child of 70 kg, 50 kg, 15 kg, and 4 kg, with the same 3 reduced static devices inserted directly into a pediatric ventilator circuit. MEASUREMENTS & RESULTS: Reduction of electrostatic charge improved small particle delivery through holding chambers to the MSLI by 12 to 14%. In the ventilator model, the mean delivery was between 1.9% and 5.4% for the nonchamber device, between 14.3% and 27.2% for the small holding chamber, and between 7.2% and 25.7% for the large holding chamber. Delivery was the least efficient in the 4-kg model compared to the 70-kg, 50-kg, and 15-kg models. CONCLUSIONS: Salbutamol from an HFA pMDI is delivered efficiently through in-line holding chambers with reduced static in pediatric ventilator settings. A large holding chamber has no advantage over a small holding chamber. In addition, salbutamol delivery is more efficient through a holding chamber than through a nonchamber device.

Preparing for Pulmonary Resection: Preoperative Evaluation of Patients—Reilly JJ. Chest 1997;112(4 Suppl):206S.

Preoperative evaluation of patients being considered for pulmonary resection is a common practice for both pulmonologists and internists. Traditionally, preoperative evaluation of this population has entailed identifying patients in whom pulmonary resection carries an unacceptably high risk of morbidity and mortality. However, recent advances in surgical technique and patient management have prompted a reconsideration of traditional preoperative approaches. This article reviews procedures currently used in the preoperative evaluation of patients considered for pulmonary resection, including the patient history, physical examination, and preoperative interventions, and addresses further evaluation of the high-risk patient.

COPD and Other Diseases in Chronically Ventilated Patients in a Prolonged Respiratory Care Unit: A Retrospective 20-Year Survival Study—Votto J, Brancifort JM, Scalise PJ, Wollschlager CM, ZuWallack RL. Chest 1998;113(1):86.

OBJECTIVES: To evaluate the long-term prognosis of ventilator-dependent patients. DESIGN: Retrospective study. SETTING: A prolonged respiratory care unit (PRCU). The PRCU provides comprehensive medical, nursing, and respiratory care to tracheostomized, ventilator-dependent adult patients who had failed all attempts at weaning. Because of their medical complexity, these patients could not be discharged to a lower level of care. PATIENTS: Of the 293 patients admitted to the PRCU over a 20-year period beginning January 1, 1977, 145 had respiratory failure from COPD, 22 from spinal cord disease or trauma, 34 from primary CNS disease, 50 from primary neuromuscular disease, and 16 from chest wall disease. Twenty-six patients were not classifiable into the above categories. MEASUREMENTS: Demographics, diagnoses, and survival data were re-

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viewed. The survival of patients with COPD was compared with the other diagnosis categories using the Cox proportional hazards model. RESULTS: The median survival for the entire group was 9 months; younger age and female gender were both predictive of longer survival (both, $p < 0.001$). The median survival of those with COPD (5 months) was significantly shorter than that of patients with spinal cord disease (47 months), neuromuscular disease (17 months), and chest wall disease (27 months) (all, $p < 0.01$). These differences in survival were present even with inclusion of gender and age in the model as covariates. The survival of patients with CNS disease was not significantly different from survival of patients with COPD. CONCLUSION: Chronically ventilated patients with respiratory failure from COPD have a significantly worse prognosis than patients with respiratory failure from other causes.

Bronchoscopic Balloon Dilatation in the Combined Management of Postintubation Stenosis of the Trachea in Adults—Noppen M, Schlesser M, Meysman M, D'Haese J, Peche R, Vincken W. Chest 1997;112(4):1136.

Bronchoscopic balloon dilatation (BBD) using angioplasty balloon catheters has been employed successfully in the treatment of tracheobronchial stenoses in children and has worked with variable success in adults with bronchial stenosis. In adults with tracheal stenosis, BBD only has been reported anecdotally. In this study, experience with BBD using a valvuloplasty balloon catheter in the combined treatment (with Nd-YAG laser photoresection and stenting) of severe benign postintubation tracheal stenoses in 3 adults is delineated. BBD was particularly successful in establishing tracheal patency when laser photoresection was contraindicated or was too dangerous; BBD allowed easy insertion of tracheal stents and the "opening" of folded silicone stents. BBD is a simple, inexpensive, safe, and efficient adjunct in the combined treatment of severe postintubation rigid tracheal stenosis in selected adults.

Effects of Long-Term Oxygen Therapy on Pulmonary Hemodynamics in COPD Patients: A 6-Year Prospective Study—Zielinski J, Tobiasz M, Hawrylkiewicz I, Sliwinski P, Palasiewicz G. Chest 1998;113(1):65.

OBJECTIVE: To investigate effects of 6 years of domiciliary oxygen therapy on pulmonary hemodynamics in a large group of COPD patients. DESIGN: Prospective longitudinal study with serial measurements. SETTING: Research institute of pulmonary diseases. PATIENTS: Ninety-five patients (72 men, 23 women), mean age 58 ± 9 years, had COPD but were free of any other serious disease. Functional characteristics at entry, mean ± SD, were as follows: FVC = 2.24 ± 0.51 L; FEV_1 = 0.84 ± 0.31 L; P_{aO_2} = 55 ± 6 mm Hg; P_{aCO_2} = 48 ± 9 mm Hg; mean pulmonary arterial pressure (PAP) = 28 ± 11 mm Hg; and pulmonary vascular resistance (PVR) = 353 ± 172 dyne · s · cm^{-5}. METHODS: Pulmonary hemodynamics were investigated using Swan-Ganz thermodilution catheters. After initial assessment, all patients were started on a regimen of long-term oxygen therapy (LTOT). Follow-up consisted of medical examination, spirometry, and arterial blood gas analysis every 3 months. Pulmonary artery catheterization was repeated every 2 years. RESULTS: Seventy-three subjects survived 2 years of LTOT. In 39 subjects catheterized after 2 years, PAP fell from 25 ± 8 to 23 ± 6 mm Hg (not significant [NS]). From 31 patients who completed 4 years of LTOT, hemodynamic data were obtained in 20. In these 20 patients, PAP averaged 24 ± 7 mm Hg at entry, and 23 ± 5 and 26 ± 6 mm Hg after 2 and 4 years, respectively (NS). In 12 patients who completed 6 years of LTOT, PAP was 25 ± 7 at entry, and 21 ± 4, 26 ± 7, and 26 ± 6 mm Hg at 2, 4, and 6 years, respectively ($p < 0.01$ for 2 vs 6 years). PVR was 313 ± 159 dyne · s · cm^{-5} at entry, and 268 ± 110, 344 ± 82, and 332 ± 205 dyne · s · cm^{-5} at 2, 4, and 6 years, respectively (NS). During 6 years of follow-up, P_{aO_2} decreased from 61 ± 3 to 46 ± 9 mm Hg ($p < 0.001$) and P_{aCO_2} increased from 44 ± 13 to 49 ± 9 mm Hg ($p < 0.01$). CONCLUSION: LTOT for 14 to 15 h/d resulted in a small reduction in pulmonary hypertension after the first 2 years followed by a return to initial values and subsequent stabilization of PAP over 6 years. The long-term stabilization of pulmonary hypertension occurred despite progression of the airflow limitation and of hypoxemia.

Impact of Clinical Pathways and Practice Guidelines on the Management of Acute Exacerbations of Bronchial Asthma—Bailey R, Weingarten S, Lewis M, Mohsenifar Z. Chest 1998;113(1):28.

OBJECTIVES: In 1990, it was estimated that approximately 1% of all U.S. health care costs (approximately $6.2 billion) were spent on asthma-related health expenses. Of this, hospitalization charges alone exceeded $2.6 billion. Practice guidelines and clinical pathways are being developed to standardize the management of acute asthma with the aim of improving care and safely reducing health care costs. In this report, we evaluate the impact of an asthma pathway developed and instituted at a large community-based teaching hospital. This pathway was evidence based and was developed by a multidisciplinary group. METHODS: The study was conducted during a 6-month period in 1995, while a similar period in 1994 was used as a historical control period. Data collected included patient demographics, hospital admission and discharge peak expiratory flow rates, pulse oximetry measurements, length of stay, conversion from hand-held nebulizer to metered dose inhaler, use of corticosteroids within 24 hours of hospitalization, and conversion of I.V. steroids to oral steroids. RESULTS: A total of 42 patients were enrolled during the study period. Of these, 19 were placed on the pathway, while 23 were not treated according to the pathway. There were 38 patients in the 1994 historical control period. For 1995, there was no significant difference between the pathway and nonpathway groups with regard to the length of stay (4.4 ± 3.3 vs 3.2 ± 2.3 days; $p > 0.05$), hospital discharge peak expiratory flow rates (324 vs 286 L/min; $p > 0.05$), or use of steroids (100% vs 91%; $p > 0.05$). However, a significant increase in conversion from hand-held nebulizer to metered-dose inhaler was noted in the pathway group (68% vs 34%; $p < 0.05$). The data from 1994 compared to 1995 pathway were similar in that there was no difference in the length of stay (3.4 ± 2.1 vs 4.4 ± 3.3 days; $p > 0.05$) and/or use of steroids (92% vs 100%; $p > 0.05$), while a significant increase in hand-held nebulizer to metered-dose inhaler conversion was observed for the 1995 pathway group (68% vs 26%; $p = 0.002$). CONCLUSIONS: We conclude that although the asthma pathway did not significantly reduce length of stay, it was associated with a significant increase in hand-held nebulizer to metered dose inhaler conversion, resulting in a substantial cost savings of $288,000/year.

Lung Transplantation in Cystic Fibrosis: Consensus Conference Statement—Yankaskas JR, Mallory GB Jr. Chest 1998;113(1):217.

The first successful heart-lung and lung transplant operations in cystic fibrosis (CF) patients were performed in 1983 and 1987, respectively. Lung transplantation is now available at dozens of centers in North America, Europe, and Australia. Recent technical developments and the major limitations of donor organ availability prompted the CF Foundation to sponsor a meeting of 37 experts to evaluate the state of the art in lung transplantation for CF, highlighting areas of consensus, practice variations, and controversy. This document summarizes the work of that group.

Evaluation of a Ventilation Strategy to Prevent Barotrauma in Patients at High Risk for Acute Respiratory Distress Syndrome. Pressure- and Volume-Limited Ventilation Strategy Group—Stewart TE, Meade MO, Cook DJ, Granton JT, Hodder RV, Lapinsky SE, Mazer CD, McLean RF, Rogovein TS, Schouten BD, Todd TR, Slutsky AS. N Engl J Med 1998;338(6):355.

BACKGROUND: A strategy of mechanical ventilation that limits airway pressure and tidal volume while permitting hypercapnia has been recommended for patients with the acute respiratory distress syndrome (ARDS). The goal is to reduce lung injury due to overdistention. However, the efficacy of this approach has not been established. METHODS: Within 24 hours of intubation, patients at high risk for ARDS were randomly assigned to either pressure- and volume-limited ventilation (limited-ventilation group), with the peak

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inspiratory pressure maintained at 30 cm H_2O or less and the tidal volume at 8 mL/kg of body weight or less, or to conventional ventilation (control group), with the peak inspiratory pressure allowed to rise as high as 50 cm H_2O and the tidal volume at 10 to 15 mL/kg. All other ventilatory variables were similar in the 2 groups. RESULTS: A total of 120 patients with similar clinical features underwent randomization (60 in each group). The patients in the limited-ventilation and control groups were exposed to different mean (± SD) tidal volumes (7.2 ± 0.8 vs 10.8 ± 1.0 mL/kg, respectively; $p < 0.001$) and peak inspiratory pressures (23.6 ± 5.8 vs 34.0 ± 11.0 cm H_2O, $p < 0.001$). Mortality was 50% in the limited-ventilation group and 47% in the control group (relative risk, 1.07; 95% confidence interval, 0.72-1.57; $p = 0.72$). In the limited-ventilation group, permissive hypercapnia (arterial carbon dioxide tension, > 50 mm Hg) was more common (52% vs 28%, $p = 0.009$), more marked (54.4 ± 18.8 vs 45.7 ± 9.8 mm Hg, $p = 0.002$), and more prolonged (146 ± 265 vs 25 ± 22 hours, $p = 0.017$) than in the control group. The incidence of barotrauma, the highest multiple-organ-dysfunction score, and the number of episodes of organ failure were similar in the 2 groups; however, the numbers of patients who required paralytic agents (23 vs 13, $p = 0.05$) and dialysis for renal failure (13 vs 5, $p = 0.04$) were greater in the limited-ventilation group than in the control group. CONCLUSIONS: In patients at high risk for ARDS, a strategy of mechanical ventilation that limits peak inspiratory pressure and tidal volume does not appear to reduce mortality and may increase morbidity.

Shifting Percussion Dullness of the Chest: A Sign of Pleural Effusion—Gilbert VE. South Med J 1997;90(12):1255.

Early physicians diagnosed pleural effusion by detecting shifting percussion dullness of the chest. However, the lateral decubitus positions have not been routinely used to distinguish this condition from atelectasis or pneumonia. In this report, postural alterations of percussion dullness established the diagnosis of pleural effusion in a 49-year-old patient with a cough. Percussion in the lateral decubitus positions may enhance the value of examinations of the chest.

The Relation of Pneumothorax and Other Air Leaks to Mortality in the Acute Respiratory Distress Syndrome—Weg JG, Anzueto A, Balk RA, Wiedemann HP, Pattishall EN, Schork MA, Wagner LA. N Engl J Med 1998;338(6):341.

BACKGROUND: In patients with the acute respiratory distress syndrome (ARDS), pneumothorax and other air leaks—any extrusion of air outside the tracheobronchial tree—have been attributed to high ventilatory pressures or volumes and linked to increased mortality. METHODS: We analyzed data from a prospective trial of aerosolized synthetic surfactant in 725 patients with the acute respiratory distress syndrome induced by sepsis. We compared the ventilatory pressures and volumes in the patients without any air leaks (the highest values during the 5-day study) with the pressures and volumes in those with pneumothorax or with any air leaks (the highest values during the 16- and 24-hour periods before the complication developed). RESULTS: Fifty patients (6.9%) had pneumothorax and 77 (10.6%) had pneumothorax or other air leaks. There were no significant differences between patients with air leaks and those without air leaks in any pressure or volume examined. Overall mortality at 30 days was 40.0% (95% confidence interval [CI], 36.4 to 43.6); among the patients with pneumothorax, it was 46.0% (95% CI, 32.2 to 59.8), and among those without pneumothorax, it was 39.3% (95% CI, 35.6 to 43.0; $p = 0.35$). The mortality rate was 45.5% (95% CI, 34.4 to 56.6) in the group with any air leaks and 39.0% (95% CI, 35.3 to 42.8) in the group without air leaks ($p = 0.28$). CONCLUSIONS: In patients with sepsis-induced ARDS who were receiving mechanical ventilation with conventional pressures and volumes, there were no significant correlations between high ventilatory pressures or volumes and the development of pneumothorax or other air leaks. Pneumothorax or other air leaks were not associated with a significantly increased mortality rate.

Effect of a Protective-Ventilation Strategy on Mortality in the Acute Respiratory Distress Syndrome—Amato MB, Barbas CS, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, Kairalla RA, Deheinzelin D, Munoz C, Oliveira R, Takagaki TY, Carvalho CR. N Engl J Med 1998;338(6):347.

BACKGROUND: In patients with the acute respiratory distress syndrome (ARDS), massive alveolar collapse and cyclic lung reopening and overdistention during mechanical ventilation may perpetuate alveolar injury. We determined whether a ventilatory strategy designed to minimize such lung injuries could reduce not only pulmonary complications but also mortality at 28 days in patients with the acute respiratory distress syndrome. METHODS: We randomly assigned 53 patients with early acute respiratory distress syndrome (including 28 described previously), all of whom were receiving identical hemodynamic and general support, to conventional or protective mechanical ventilation. Conventional ventilation was based on the strategy of maintaining the lowest positive end-expiratory pressure (PEEP) for acceptable oxygenation, with a tidal volume of 12 mL/kg of body weight and normal arterial carbon dioxide levels (35 to 38 mm Hg). Protective ventilation involved end-expiratory pressures above the lower inflection point on the static pressure-volume curve, a tidal volume of < 6 mL/kg, driving pressures of < 20 cm H_2O above the PEEP value, permissive hypercapnia, and preferential use of pressure-limited ventilatory modes. RESULTS: After 28 days, 11 of 29 patients (38%) in the protective-ventilation group had died, as compared with 17 of 24 (71%) in the conventional-ventilation group ($p < 0.001$). The rates of weaning from mechanical ventilation were 66% in the protective-ventilation group and 29% in the conventional-ventilation group ($p = 0.005$): the rates of clinical barotrauma were 7% and 42%, respectively ($p = 0.02$), despite the use of higher PEEP and mean airway pressures in the protective-ventilation group. The difference in survival to hospital discharge was not significant; 13 of 29 patients (45%) in the protective-ventilation group died in the hospital, as compared with 17 of 24 in the conventional-ventilation group (71%, $p = 0.37$). CONCLUSIONS: As compared with conventional ventilation, the protective strategy was associated with improved survival at 28 days, a higher rate of weaning from mechanical ventilation, and a lower rate of barotrauma in patients with ARDS. Protective ventilation was not associated with a higher rate of survival to hospital discharge.

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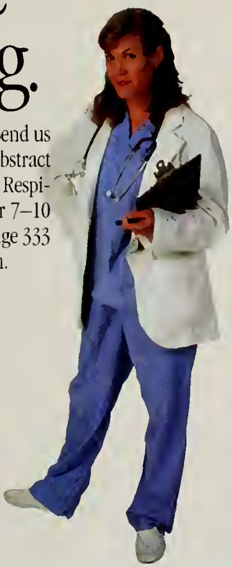

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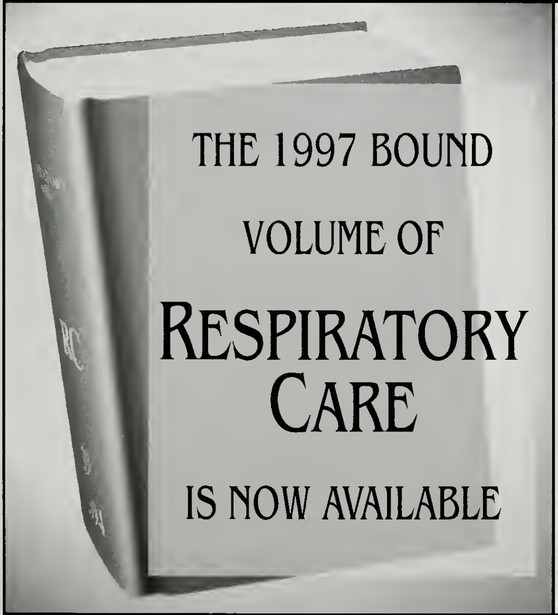

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American Respiratory Care Foundation

— Foreword —

Susceptibility, Strategies of Care, and Schooling in Sleep-Disordered Breathing: The 1997 RESPIRATORY CARE Journal Conference

Sleep-disordered breathing is a common and important clinical problem, estimated to affect 2-4% of Americans and associated with significant adverse consequences such as cardiovascular morbidity and early demise. Because major advances in both diagnosis and management have been made in the recent past and because respiratory care practitioners are participating in the care of these patients, the RESPIRATORY CARE Journal Conference Special Issues address Sleep-Disordered Breathing (SDB). In keeping with Journal Conference tradition, the April and May 1998 issues present the papers that accompanied each of the presentations made during the Conference (conducted on October 17-19, 1997) and the edited transcripts of the spirited discussions that followed each presentation.

The faculty consisted of a group of expert respiratory care practitioners and physicians, invited because of their knowledge and important contributions to this field. (In the case of the three authors of this foreword, the alliteration of their last names was a coincidental bonus).

The three goals of the Journal Conference are addressed in the pages that follow:

- To review the current state-of-the-art in diagnosis and management of sleep-disordered breathing in both adults and children.
- To highlight the emerging role of respiratory care practitioners in the care of patients with sleep-disordered breathing.
- To generate these issues of RESPIRATORY CARE to present this important information to the respiratory care community.

In addressing these three goals, the Journal Conference chairmen organized 12 topics and the Conference summary into 4 categories: Susceptibility Issues regarding Sleep-Disordered Breathing, Diagnosis and Treatment of SDB in Adults, Diagnosis and Treatment in Children, and the Delivery of Clinical Services to Manage Sleep-Disordered Breathing. The 12 faculty members represented 10 institutions in 9 geographically diverse states representing the East, Midwest, South, and West.

As noted by Dr Strollo in the Conference Summary, several important general points emerged from the presentations and discussions.

First, the burgeoning information about the epidemiology and pathophysiology of sleep-disordered breathing requires a careful re-consideration of whom we should treat and why. The results of ongoing multicenter longitudinal, population-based studies are eagerly awaited to enhance the strategy of care with better diagnostic criteria, recognition of important clinical sequelae, and ability to predict the natural history of SDB. Second, new and evolving medical and surgical treatments expand the spectrum of therapeutic options while posing continued challenges to clarify their efficacy and relative roles. Finally, proper diagnosis and management of patients with sleep-disordered breathing require close interaction between sleep physicians and well-trained, credentialed allied health care colleagues. By virtue of their technical expertise, respiratory care practitioners are well-positioned to gain special expertise and credentials in sleep medicine and can contribute importantly to clinical and investigative needs.

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Pathogenesis of Obstructive Sleep Apnea

Alejandro D Chediak MD

Introduction
The Upper Airway
  Anatomy
  Pressure Relationships
  Neuromuscular Influences
  Other Factors Affecting Patency
    Anatomic Factors
    Gender
    Posture & Gravity
    Surface Adhesive Forces
    Vascular Factors
    Loss of Post-Stimulus Potentiation
    Circadian Rhythms of Hormones & Cytokines
    Nasal Influences
Summary

Introduction

Early investigators suspected the pharynx as the site of upper airway (UA) obstruction in obstructive sleep apnea (OSA), a finding that was supported by the observation that tracheostomy could reverse the abnormal physiology and symptoms in patients suffering from this disorder.[1-5] It seems intuitive that neural mechanisms are involved in pathogenesis of OSA as the disorder is intimately linked to sleep. However, the clinical observation that obesity is the dominant factor in the appearance of OSA in men and women suggests that anatomic factors explain UA closure in OSA.[6] We currently possess only fragmentary knowledge of the abnormalities that underlie closure of the UA in patients with OSA. Nonetheless, the available evidence indicates that in OSA the site of UA obstruction is the pharynx and that obstruction of the pharynx during sleep is a result of an imbalance between the forces that serve to dilate the pharynx and those that promote pharyngeal closure.[7-11] In this article I review the factors that have been implicated in the pathogenesis of UA closure during sleep (Table 1).

The Upper Airway

Anatomy

The UA begins at the nasal and oral orifices and terminates at the glottic aperture (Fig. 1). Airway occlusion in OSA can occur at single or multiple sites between the soft palate and the epiglottis. Narrowing of the UA most commonly occurs at the soft palate, but there may be considerable variability.[12] In some patients with OSA, UA occlusion occurs at more than one site and the site of airway collapse can move caudally during the transition from non-rapid-eye movement (NREM) to rapid-eye-movement (REM) sleep.[13]

Pressure Relationships

The so-called balance of pressure theory considers the UA as a collapsible tube in which UA transmural pressure (P_{tm})

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Table 1. Factors Implicated in the Pathogenesis of Obstructive Sleep Apnea

- Pharyngeal neuromuscular considerations
  - Sleep-induced alterations in respiratory muscle drive
  - Reflex alterations in respiratory drive
  - Increased pharyngeal compliance
  - Disproportionate contraction of respiratory muscles
  - Delayed onset of upper airway muscle contraction
  - Sleep-deprivation effects on upper airway motor tone
- Pharyngeal anatomic considerations
  - Obesity
  - Macroglossia
  - Craniofacial structural abnormalities
- Miscellaneous factors
  - Effect of gender
  - Posture and gravitational influences
  - Surface adhesive forces
  - Vasomotion
  - Loss of short-term post-stimulus potentiation
  - Alteration of cytokine circadian rhythm
  - Nasal obstruction

is defined as the difference between luminal (P_l) and tissue pressure (P_{ti}) and where P_{ti} may not always equal atmospheric pressure. Accordingly, increases in P_{tm} serve to augment the cross-sectional area of the passive UA as predicted by a P_{tm}-area relationship termed the tube law of the UA (Fig. 2). In the absence of airflow, P_l is equal to atmospheric pressure. Inspiration is accompanied by a reduction in P_l due to the dissipation of energy to overcome upstream resistance and because of acceleration of gas flowing through a narrowed airway. P_{ti} is influenced by forces acting on the outside surface of the pharyngeal walls, such as neck compression, excessive submandibular fat, and macroglossia—all of which may potentially augment P_{ti}. The relationship between P_{tm} and UA cross-sectional area is curvilinear and as such its slope (dA/dP_{tm}), or compliance, varies with luminal aperture (Fig. 2). A similar relationship between pressure and area has been demonstrated in the hypotonic pharynx of humans with OSA.[14] Activation of UA-dilating muscles increases luminal area for a fixed P_l and P_{ti} and, thereby, shifts the P_{tm}-cross-sectional area curve to the left with the maximal effect seen near the UA closing pressure (Fig. 2). Sleep-induced alterations of UA muscular forces and declines in lung volume serve to enhance UA collapsibility and facilitate inspiratory flow limitation and periodic obstructive apnea and hypopnea (Fig. 3).

Neuromuscular Influences

We now recognize that pharyngeal patency is attributable in part to behavioral influences. The central nervous system coordinates the UA aperture during swallowing and speech in such a manner as to allow only transient airway closure. Flow of air into the lungs requires coordinated activation of UA, thoracic, and diaphragmatic muscles. Analysis of UA and diaphragmatic electromyographic (EMG) activity during inspiration demonstrates UA activation that precedes diaphragmatic activity by 0.2 seconds.[15,16] Contraction of UA-dilating muscles before inspiratory airflow serves to stiffen and increase the aperture of the UA and prepare it for the suction forces that accompany inspiration. In healthy awake subjects, muscular relaxation during negative pressure breathing leads to a reduction in size or closure of the UA, presumably due to an uncoupling of UA and diaphragm muscular activity.[17]

The genioglossus, geniohyoid, sternohyoid, tensor palatini, and medial pterygoid muscles tend to produce ventral movement of the tongue, soft palate, and mandible when activated during the inspiratory phase of breathing.[8,18] In anesthetized dogs, stimulation of the hypoglossal nerves improved UA patency, both by dilating the UA and stiffening its walls.[19] Submental electrical stimulation of UA muscles has been shown to abbreviate and reduce in number obstructive apneas and hypopneas in humans with OSA, but the effect appeared to be mediated through arousal from sleep.[20] Chemical and nonchemical factors can contribute to recruitment of an isolated UA muscle, such as the genioglossus.[21,22] The precise contribution to UA patency afforded by the contraction or relaxation of an isolated UA muscle is currently not known. Contraction of UA-dilating muscles shifts the relationship of P_{tm} and cross-sectional area to the left (Fig. 2).[23] However, it is not possible to estimate the magnitude of change in dA/dP_{tm} because the muscles are interdependent and modifications of head and mandibular posture alter the length-tension relationship, thereby modifying the muscular force produced by a specific amount of neural activation.[24,25] During sleep, tonic and phasic respiratory activity of the UA muscles is somewhat reduced and dA/dP_{tm} increases,[26,27] in contrast to diaphragmatic activity, which appears to be preserved in normal sleeping humans and in those with OSA.[28] The net result favors pharyngeal narrowing during sleep. As UA aperture declines, a further loss in P_l occurs due to the conversion of potential energy to kinetic energy as the velocity of gas increases when it passes through the narrow airway segment. The resultant loss in muscular forces and P_l eventually leads to inspiratory flow limitation and/or complete UA occlusion.

Mathew and co-workers[29,30] have demonstrated augmentation of UA motor output in response to decreasing intrapleural pressure. Elimination of vagally-mediated volume feedback produced an increase in genioglossal neural output in decerebrate cats.[30] These reflex responses potentially can be elicited in response to UA occlusion and might play a role in termination of apnea and hypopnea during sleep.

Snoring or other injury to the UA associated with repetitive airway closure may lead to a local neuropathy and facilitate the development of OSA.[32] Once the UA is occluded, arousal from sleep is usually required to restore patency.

Sleep deprivation per se can suppress UA motor function during sleep.[33] In general, pharmacologic agents that inhibit

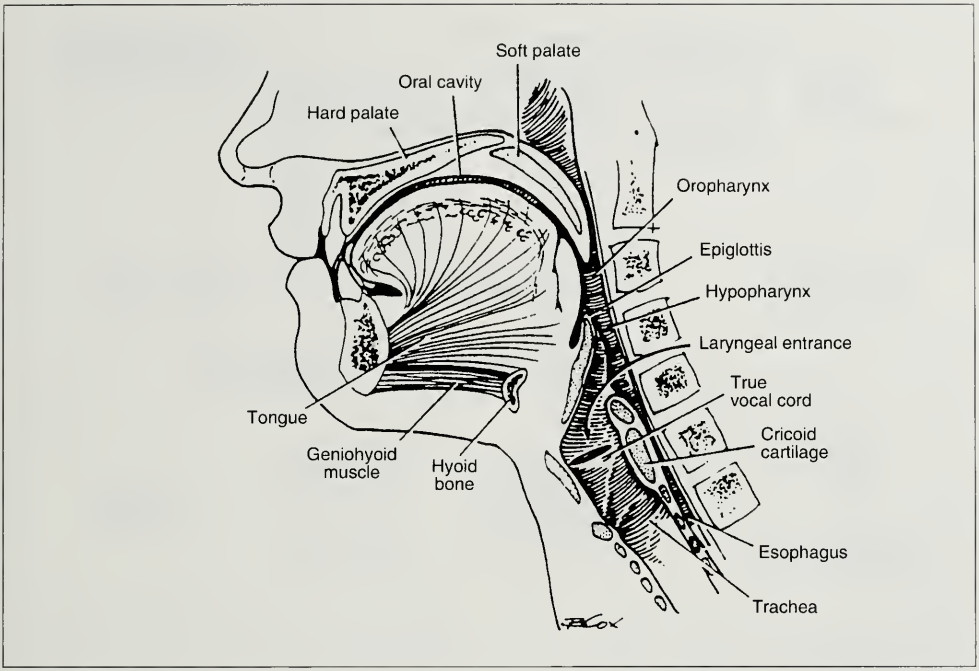

Fig. 1. The anatomy of the upper airway.

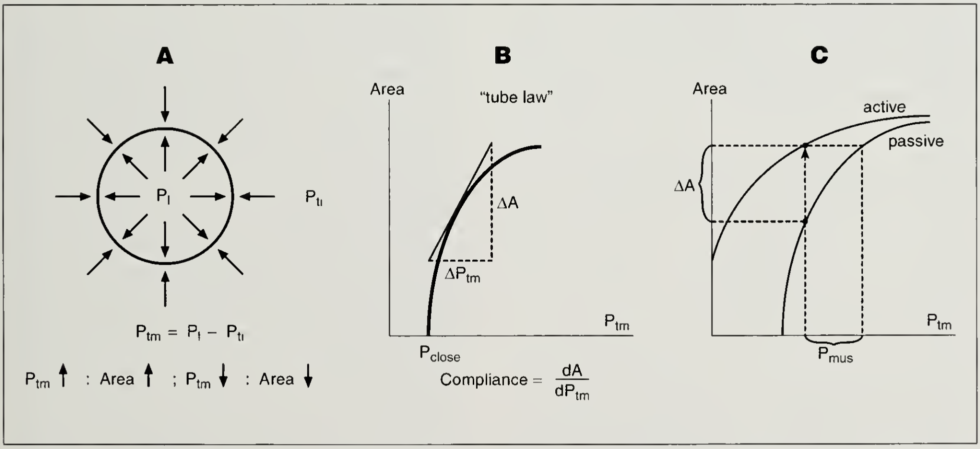

Fig. 2. Illustrations of the balance of forces theory and the tube law of upper airway stability. **A.** Transmural pressure (P_{tm}) is defined as intraluminal pressure (P_l) minus surrounding tissue pressure (P_{ti}). **B.** The relationship between P_{tm} and the collapsible tube's cross-sectional area (A), with compliance defined as the slope of this relationship (dA/dP_{tm}). **C.** Contraction of upper airway dilators augments cross-sectional area A, resulting from a modification of the tube law and while P_{tm} remains constant. Reprinted from Reference 23, with permission .

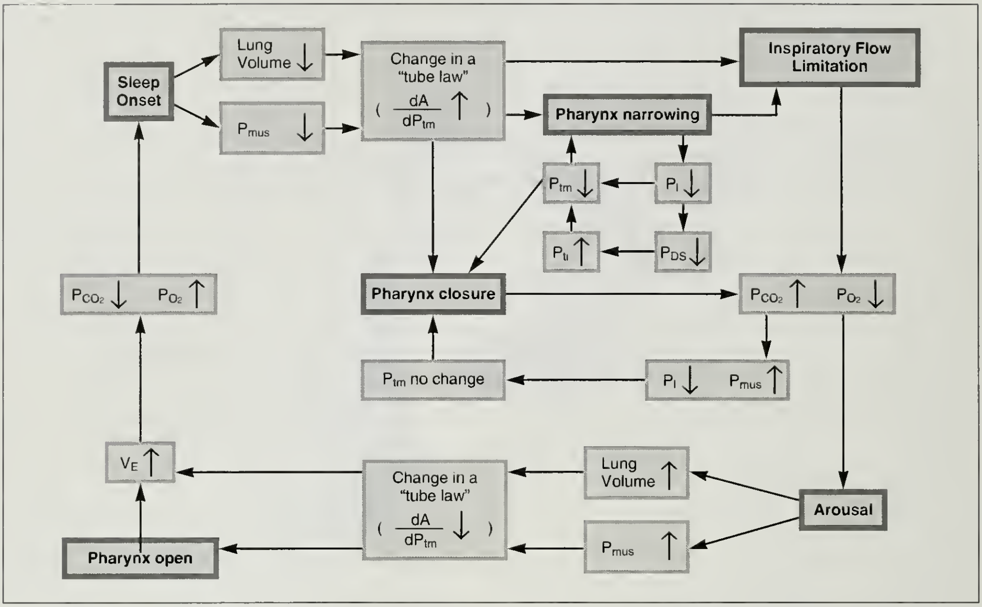

Fig. 3. The sequential events leading to periodic upper airway occlusion-ventilation (V_E) in OSA. P_{mus} = muscular pressure; dA/ dPt_m = compliance of the upper airway; P_I = intraluminal pressure; P_{ti} = surrounding tissue pressure; P_{DS} = downstream pressure. Reprinted from Reference 23, with permission.

central nervous system activity tend to depress motor output to UA muscles, such as the genioglossus.[34] Topical anesthetics applied to the UA can increase the frequency of sleep-related obstructive apneas and hypopneas, perhaps by a reflex mechanism.[35] A list of commonly used pharmacologic agents that are known to facilitate UA occlusion during sleep appears in Table 2.

Table 2. Agents that Can Worsen Obstructive Sleep Apnea

Ethanol
Opiates
Benzodiazepines
Imidazoperidines
Systemic anesthetics
Topical anesthesia

The notion that behaviorally-dependent neuromuscular mechanisms play an important role in OSA is exemplified by the observation that UA occlusion in OSA occurs solely during sleep. However, it is not known at this time (1998) whether patients with OSA exhibit greater than normal sleep-related depression of UA neuromuscular activity.

Other Factors Affecting Patency

Anatomic Factors. The notion that anatomic factors influence UA patency in OSA is supported by the observation that gains in body weight tend to increase and losses of body weight lessen the frequency of obstructive apneas and hypopneas during sleep.[36-41] The UA area enclosed by the mandible ramus as well as the volume of adipose and muscular tissue surrounding the UA of patients with OSA correlates with the frequency of apneas and hypopneas.[41-43] Weight gain may further compromise UA patency by increasing the weight and bulk of the tissues surrounding the pharyngeal lumen and elevating P_{ti}.[44] The collapsibility of the UA may be indirectly influenced by obesity-related declines in lung volume and changes in central venous pressure.[45,46] The cumulative effects of increasing body weight on UA function serves to facilitate closure of the passive airway, either by elevating P_{ti} or altering dA/dP_{tm}.

Sullivan and co-workers[47,48] measured UA collapsing pressure during sleep in normal subjects, habitual snorers, and patients with OSA and found that normal humans could resist up to −15 cm H_2O pressure before UA closure occurred. In patients with OSA, closing pressures during sleep were usually less than −3 cm H_2O pressure and were often positive.

The differences observed in collapsing pressure can be explained theoretically by alterations of UA structure and suggest that the closing pressure of the passive UA of humans with OSA is greater than normal. Brown et al[49] examined the pressure-area relationship of the pharynx in 13 OSA patients and 7 control subjects while they were awake. Specific pharyngeal compliance was 0.036 mL/cm H_2O in controls and 0.094 mL/cm H_2O in patients with OSA. Similar findings have been obtained by other investigators.[47,49-52]

Abnormalities of UA structures are commonly found in patients with OSA.[9,53,54] Such abnormalities can be functionally demonstrated by standard and tidal-breath flow-volume curves, which may show rapid flow oscillations, variable extrathoracic obstruction, and in the case of tidal-breath curves, a posture-related change in the terminal portion of the expiratory flow-volume curve in patients with OSA.[55-60] Cine-computed tomography (CT) of the UA was used to record the effect of respiration on UA caliber in normal human subjects, mild snorers, and patients with OSA.[61] The investigators demonstrated smaller UA size and a greater tendency to airway closure at the end of expiration in patients with OSA. Although these observations are consistent with the notion of structural abnormalities as the primary defect in OSA, the data were obtained in patients during wakefulness, and therefore muscular factors were not controlled. The differences observed between OSA and normal subjects could be explained by structural or neuromuscular factors. Because patients with OSA have supranormal activation of UA-dilating muscles while awake, one would predict even greater differences in waking UA size after controlling for muscular factors.[62]

Cephalometric studies have demonstrated craniofacial abnormalities in most patients with OSA.[63] The length of the soft palate, the position of the hyoid bone, and the size of the posterior airway space are of particular interest in OSA. Using magnetic resonance imaging, Shelton and co-workers demonstrated a significant correlation between the frequency of apneas and hypopneas during sleep and the area enclosed by the mandible ramus and the distance between the teeth and the posterior mandible ramus.[42] In addition, subjects with OSA had a collection of adipose tissue adjacent to the UA. The volume of pharyngeal adipose tissue correlated with the frequency of apneas and hypopneas and weight-loss-related improvement in OSA was accompanied by a reduction in the volume of adipose tissue adjacent to the UA.[41] Several other techniques have demonstrated smaller pharyngeal areas in patients with OSA than in control subjects who were not weight-matched.[64-67] However, one study failed to demonstrate a significant difference in airway size between OSA patients and control subjects who were matched for age and weight and in whom OSA had been excluded by prior polysomnography.[68]

Sleep onset is accompanied by a rise in supraglottic resistance, even in normals.[28,69,70] Supraglottic resistance during wakefulness is greater in patients with OSA than in normal controls.[69] Advanced age has a significant correlation with increasing pharyngeal resistance in men but not in women.[71] Sleep-disordered breathing is more common in groups in which pharyngeal resistance is reportedly elevated, such as the obese, the elderly, and in men.

In summary, the existing evidence supports the notion that structural abnormalities of the UA are present in patients with OSA and that alterations of UA anatomy play a major role in the pathogenesis of UA occlusion during sleep. The degree to which anatomic factors contribute to OSA cannot be determined precisely, partly because the available data have been collected on subjects who were awake and without controlling for muscular factors.

Gender. Although OSA is more prevalent in men, healthy women have smaller pharyngeal areas than healthy men during wakefulness.[72] However, men were shown to have a larger change in pharyngeal area relative to lung volume, a factor consistent with the greater incidence of OSA in men. Guilleminault et al[6] compared selected clinical features of women and men with OSA who had been matched for the frequency of sleep apneas and hypopneas and craniofacial structure by cephalograms and found that the women with OSA were much more obese than their male counterparts suggesting superior protection of UA patency during sleep by women (Table 3).[6] In awake, healthy women, Popovic and White[73] demonstrated elevated genioglossal activity with quiet breathing and in response to inspiratory loading as compared to healthy awake men. It appears that the female UA may be more stable and better suited to withstand collapsing forces that accompany sleep.

Table 3. Comparison of 27 Women with Obstructive Sleep Apnea to 27 Men Best-Matched for Age, Respiratory Disturbance Index (RDI), and Cephalometric Variables

Variable	Women	Men	p value
Age in years	48.5 (12.4)*	48.7 (13.0)	NS
RDI—number of apneas & hypopneas/h of sleep	63.2 (31.1)	61.5 (28.1)	NS
Posterior airway space in mm	4.8 (2.1)	5.0 (2.3)	NS
Mandibular-plane-to-hyoid length in mm†	25.5 (7.6)	24.7 (4.5)	NS
Body mass index in kg/m²	37.7 (7.4)	30.8 (4.5)	< 0.001

*Values are mean (SD); NS = nonsignificant.

†As described in Reference 62.

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Posture & Gravity. Patients with OSA often have more apneas and hypopneas when recumbent than while in other postures.[74,75] Gravity and posture also play a significant role in sleep-related increase in supraglottic resistance and in awake

UA motor tone.[22,76] In habitual snorers, the rise in supraglottic resistance may increase to values > 50 cm $H_2O \cdot s \cdot L^{-1}$.[77] The resulting decline in P_l promotes further airway collapse. Using a diaphragmatic EMG, Takasaki and colleagues[76] estimated the effect of gravity on UA resistance aboard a short-term space mission and concluded that gravity played a greater role than UA muscle atonia in sleep-related elevation of UA resistance.

Surface Adhesive Forces. UA closure is accompanied by surface adhesive forces between the opposed UA walls. Surface adhesive forces must be overcome to reopen the collapsed UA. The observation that the pressure required to reestablish airway patency exceeds closing pressure may be partially explained by surface adhesive forces.[48]

Vascular Factors. Pharyngeal vasomotion may modify in UA mechanics and, thereby, influence obstructive breathing during sleep.[78] Measurement of UA perfusion in asleep humans is technically difficult and has not been reported. However, in awake, normal humans, pharmacologic agents shown to act as UA vasodilators and vasoconstrictors fail to alter the static pressures volume relationship of the UA.[79] Nonetheless, dynamic UA properties may be influenced by topically applied vasoconstrictors.[80]

Loss of Post-Stimulus Potentiation. Abrupt cessation of respiratory stimulation is accompanied by a gradual reduction to pre-stimulus levels and does not undershoot, a phenomenon termed post-stimulus potentiation. In some OSA sufferers, post-stimulation potentiation to brief hypoxia is blunted, a condition that may, in theory, destabilize respiration and facilitate apnea.[81]

Circadian Rhythms of Hormones & Cytokines. Investigations into circadian cytokine release in OSA patients demonstrate s significant disturbance in the secretion of tumor necrosis factor-a (TNF).[82] Circadian variations in interleukin-1, interleukin-6, γ-interferon, and the hormones cortisol and melatonin did not differ between controls and subjects with OSA. Three months of therapy with nasal continuous positive airway pressure failed to normalize TNF rhythms, suggesting that the cytokine TNF may be involved in the pathophysiology of OSA.

Nasal Influences. Nasal airflow may have a stimulant effect on respiration during sleep, and nasal resistance increases while flow decreases progressively during NREM sleep.[83,84] Nasal abnormalities, such as septal deviation, are known to induce or exacerbate sleep-disordered breathing.[9,85-87] Surgical correction of nasal obstruction has been demonstrated to improve sleep-disordered breathing in selected cases. In order to overcome upstream nasal obstruction, energy is dissipated resulting in a decline of P_l that serves to promote pharyngeal closure. Nasal airway resistance is higher than oral airway resistance in normal awake humans. Observations by Olsen and associates[88] in sleeping humans suggest that oral airway resistance may exceed nasal airway resistance, possibly because the nasal airway is rigid and hence its caliber is not dependent on neuromuscular factors.

Summary

The cycle of events detailed in Figure 3[23] illustrates the potential relationship between behavioral state and the collapsible human upper airway. The currently available data support the notion that the periodic airway occlusion occurring in the sleep of patients with OSA can be explained by alterations in upper airway anatomy that facilitate upper airway occlusion during sleep and that perturbations of upper airway neuromuscular control may participate in sleep-mediated closure of the upper airway. The combined effect of ab normal structure and function in the upper airway of subjects with OSA serves to alter the balance of forces affecting the compliant upper airway, such that collapsing forces exceed distending forces.

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Discussion

Brooks: One of your early slides showed the pressure in portions of the pharynx, nasopharynx, hypopharynx, oropharynx, and so forth. The pressures in the nasopharynx were the only ones that went positive at any time. Why was that?

Chediak: That was the slide by Shepard[1] (Fig. 1).

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Brooks: If you have expiratory flow, shouldn't the pressure be positive everywhere?

Strohl: Only up until the flow is obstructed.

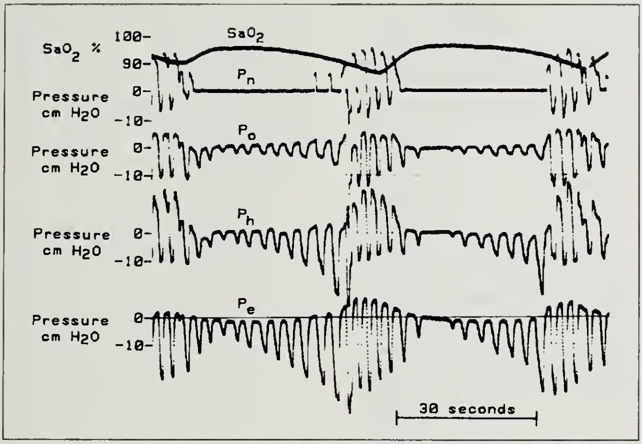

Fig. 1. Results of monitoring of pressures (during overnight polysomnography) in the posterior nasopharynx, oropharynx, hypopharynx, and esophagus in 18 male patients with moderately severe sleep apnea. Modified from Shepard JW Jr, Thawley SE. Localization of upper airway collapse during sleep in patients with obstructive sleep apnea. Reprinted from Am Rev Respir Dis 1990;141(5 Pt 1):1350-1355, with permission.

Chediak: Yes.

Brooks: OK. Now go to the next one. In this one you said that the flow limiting segment was between the ...

Chediak: Oropharynx and hypopharynx.

Brooks: So, if Kingman (Strohl) is right, we'd expect to see the oropharynx go positive, but it doesn't. Even during regular breathing, the oropharynx does have some reversal here. You see? Here's zero.

Chediak: Yes. I see.

Strohl: You have an expiratory puff.

Chediak: Right. Up there.

Strohl: Two expiratory puffs right there. So the first apnea, end of the first apnea, those were the two little puffs. Little expiratory puffs.

Brooks: Even during the unobstructed breaths in the middle, I'm seeing fairly positive pressure swings in the nasopharynx.

Chediak: Here? Well, when you reverse flow, you're going to reverse the pressure.

Brooks: Sure. OK. The other one was in the studies of hypoglossal and phrenic nerve activity. The hypoglossal activity took place a little bit before the diaphragm activation.

Chediak: Yes.

Brooks: How fast does the activation of the nerve transmit to actual pressure developed by the muscle? Since the diaphragm is a big muscle, if you stimulate that and you stimulate a small pharyngeal muscle at the same time, is there more of a lag between the response of the diaphragm and of the pharyngeal muscles?

Chediak: I can't answer the specific question, but I can tell you that the electrical activity precedes activation—the electrical activity reaching the diaphragm by about 0.2 s. There's a very short period of time where you have some activation in the upper airway dilators, none in the phrenic nerve. Now, the phrenic nerve is longer, so, theoretically, transit time is longer, and, as you suggested, the muscle is bigger. Now whether the bigger muscle translates into slower response, I don't know. The pressure generated is related also to the length/tension relationship at any given moment, so force generation is partially dependent, in terms of the diaphragm, on lung volume and, in terms of the upper airway, on neck posture, as well as general posture of the patient.

Phillips: I think the frontier of physiology for sleep-disordered breathing in the next few years may well be the nose. This is one of the tersest, most understandable summaries of the physiology of the posterior air space that I've heard. But it's amazing how little we know about the role of increased upstream resistance, as others have called it, in facilitating or maybe even causing this disorder. Some of the earliest reports from Guilleminault[1] did show an increased risk of sleep-disordered breathing in people who had deviated nasal septa, and now we have a very good report showing that nasal congestion with allergies is a significant risk factor. It's a hard thing to measure.

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Chediak: Nasal resistance is actually easy to measure during both sleep and wakefulness. It just requires measuring anterior nasal pressure and posterior pharyngeal pressure with a straw-like device you place through the lips in the back of the tongue and a nasal mask and a flowmeter. The problem with the nose—and I understand the role of upstream resistance—is that it reduces downstream luminal pressure, but remember that the nose itself is supported by a semirigid cartilaginous structure and as such is not collapsible. The upper airway distal to the supported nasal structures is far more likely to collapse. That's one of the reasons why the area of greatest interest, at least for me, is the most collapsible segment from the posterior nasal spine on below until you get right into the larynx, where you have a strong cartilaginous support system, which will not readily collapse.

Phillips: But I think that increased upstream resistance promotes collapsibility of the part that is collapsible.

Chediak: Oh, absolutely. The upstream resistance reduces luminal pressure for a given breath, and that facilitates closure. It changes the transmural pressure in the collapsible portion of the tube. The higher the upstream resistance, the more likely you are to have downstream collapse in a given individual.

Strohl: This is a terrific model, especially with the use of the 'tube law,' which has been applied to the lung for a long, long time. I guess the controversy that you're speaking to is How does the upper airway collapse? Does it collapse as a concentric tube collapses? Or is it a chock-block collapse? Also, do you think that the mechanisms that collapse the airway and cause it to close are just operating in reverse to reopen the airway?

Chediak: In terms of the manner in which the airway collapses, most of the studies suggest that it does so in a lateral fashion, not in an anterior-posterior fashion, but that's not the important question that you're asking. What happens when you look at it longitudinally, going down the tube? What Remmers's work shows[1] when he did direct visualizations is that the airway actually has various sites that may collapse; and he has suggested that if you modify one segment, the downstream segment may collapse. That was also illustrated indirectly by John Shepard's work[2] in the multiple catheter study in which those patients who had isolated oropharyngeal collapse went on to fail uvulopalatopharyngoplasty. Why did they fail? On repeat study Shepard demonstrated caudal displacement of the flow-limiting segment and hypopharyngeal collapse. Simply by modifying the mechanical properties of one area, you alter the downstream pressures and you may alter the collapsibility of the downstream segment. So, that's one of the difficulties in understanding and treating obstructive sleep apnea with a site-specific modality. There is no one site in every patient or even in a given patient that always obstructs every time. It may be one site most of the time, and, in fact, if you'll look at Remmers's work with direct visualization, a number of sites could obstruct in a given patient on a given night. Most patients with obstructive sleep apnea were demonstrated to have more than one site of obstruction. Now, do the forces simply reverse when they open up? I think the data support that the main reason the pharynx opens is because the upper airway is stimulated by arousal. Its neural input is stimulated sufficiently to dilate or to force opening. Is that a reversal of what happens during sleep? Yes, I would think so. Now, does it happen exactly in the reverse fashion? I don't know.

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Strohl: Perhaps the muscles needed to reopen the airway are different from those that permit it to close, which is why you have to have arousal (to activate other muscles). Once the airway is closed, you have apposition of mucosal forces, you have surface tensions, and it's like trying to take apart 2 pieces of glass that are wet. You can't just pull them apart. While you can let them slap together, you have to slide them apart. So, even at a mechanical level, I guess the idea is if you can't reopen the airway, you have a 12-second apnea. If you can reopen the airway in < 10 s, we won't have a syndrome anymore, as our definition of apnea begins at 10 s.

Woodson: Early in your talk, you dismissed constrictor muscles as playing much of a role in airway closure. Dempsey and Badr have looked at active airway closure with central apnea.[1] We can't ignore what happens with the constrictor muscles or what happens during expiration. Collapse is not something that occurs only during inspiration. There are major events during expiration.[2] In sleep apnea significant airway closure may occur. Airway collapse is a complex and difficult problem. We're looking at an upper airway that's unstable—this long collapsible tube that is hugely important to the organism. There is hardly any physiologic function that's less important than maintaining a patent airway, and so I think we have to expect that it's going to be hugely redundant in developing mechanisms to maintain this patency. Abnormalities in any of these may contribute to collapse.

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Chediak: Expiratory obstruction in sleep apnea has been demonstrated in a number of different ways. Whether it's due to active forces generated by constrictors is really, I think, an unanswered question. The most recent work I read was by Kuna.[1] He suggested that if you look at one isolated constrictor muscle (the superior pharyngeal constrictor), it doesn't necessarily contribute either to opening or to closing the pharynx. Now whether that translates into all the others, or a coordination of muscles, or a variety of different on/offs, if you will, one muscle turns off, another one turns off slightly, and that might alter the mechanical properties of the pharynx in facilitated closing and opening. It hasn't even been investigated, but in terms of isolated muscle investigations, with constrictors of the upper airway, I believe that there are numerous unanswered questions with regard to the role they play in pharyngeal closure and reopening. Nonetheless, as isolated muscles, they're probably not as important as the dilators.

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1. Kuna ST, Smickley JS. Superior pharyngeal constrictor activation in obstructive sleep apnea. Am J Respir Crit Care Med 1997;156(3 Pt 1):874-880.

Atwood: I am curious about your study on upper airway blood flow and the issue of whether the upper airway is edematous or not. Was your study done in normal or in apneic patients?

Chediak: Normals.

Atwood: I think many of us who look in the back of patients' throats on a daily basis are struck by how edematous the airway looks. Do we have good data that show that, in fact, patients with sleep apnea have edematous airways? It looks edematous to us, but how can we prove that?

Chediak: Well, there are data from MRI [magnetic resonance imaging] studies, looking at tissue mucosal volumes in patients with sleep apnea,[1,2] and they do appear to have a reversible component of tissue engorgement—reversible meaning that you treat them with CPAP for a number of weeks and the tissue gets somewhat thinner. In my laboratory, we haven't looked at perfusion, per se, in sleep apneics yet. So, I can't directly answer the question Do sleep apneics have a hyperperfused upper airway? Another question, Is it flow through the tissues or is it the amount of blood in the tissues? Because those blood vessels, in theory, could congest permanently and increase the total blood volume, and if you increase the total blood volume in the tissue, but not necessarily the rate of blood flow to the tissues, then you have a heavier structure, and for a given amount of neural activation, that structure becomes somewhat more collapsible. Any manipulation that would lead to heavier, bulkier upper airway structures would increase supraglottic resistance and would serve to reduce luminal forces for a given breath and facilitate closure during sleep.

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Strollo: On the issue of airway closure and vascular congestion, it's interesting that the patients with congestive heart failure actually have an increased incidence of obstructive apnea that Bradley has identified.[1] It may play a role in vascular congestion in upper airway structures. That's been postulated recently, and some folks are looking at that. One of the questions with regard to pathophysiology that we didn't touch on is airway damage. In Edinburgh recently some of the data that evolved was that there may be pressure receptors that contribute to airway damage in muscle and nerves, in the palate at least. It may explain in part the conundrum—many people present with snoring at one point, but it takes a period of time to develop obstructive sleep apnea. Airway damage doesn't have much of an impact on treatment with positive pressure when we're splinting the airway opening, but I think it may have an impact when one operates on somebody. You may enlarge the airway, but the response to changes in compliance may be permanently altered. Would you like to comment on that?

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Chediak: Yes, I can. There is one paper that I'm aware of that looks at sensation in the pharynx of sleep apneic patients versus normals.[1] The investigators stimulated the pharynx with heat and cold and a variety of distasteful stimuli and looked at the responses in sleep apneics versus subjects who were not sleep apneics. They suggested that there's a local neuropathy of the pharyngeal structures in patients with obstructive sleep apnea, at least in the soft palate. Pharyngeal neuropathy might translate into defects in sensors whether they be barosensors or temperature sensors or other sorts of upper airway sensors that would put the upper airway at risk of closure during sleep. So, there is some evidence that the upper airway is affected and perhaps damaged, as you suggest, in a manner that would facilitate closure during sleep. Now whether that damage is due to snoring, I'm not certain. As you know, at Mount Sinai a number of years ago Dr Martin Cohn looked histologically at the soft palate of snorers with OSA who underwent uvulopalatopharyngoplasty and described changes that were suggestive of what one sees with vibration injury.[2] So, there is evidence of structural and sensory abnormalities in the soft palate of patients with OSA. Certainly that could be a manifestation of airway injury over a number of years, either by vibration, contact with walls, or by suction forces.

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2. Cohn M, Hesla PE, Kiel M, Nay KN. Vibration frequency of snoring in obstructive sleep apnea syndrome (abstract). Chest 1986;89(6 Suppl):529S.

Minkley: I've been impressed with the number of variables involved even when we look at something simple like the anatomic efforts to breath through a tube versus a structure (the upper airways and trachea) in which there are several opportunities for obstruction, and the difficulties in having to study and investigate those aspects to determine areas of obstruction that will ultimately determine treatment options and help develop technology to investigate those aspects. There are added difficulties in the sleeping human—How do we look at them and maintain normal physiologic sleep? What are the differences in cardiorespiratory physiology in REM versus non-REM sleep? What are the differences in physiology, as you pointed out, in a sleep-deprived patient versus a well-slept normal sleeping patient. While in animal models we can induce sleep, these variabilities seem really overwhelming to try to get an answer to what at first seems like a simple question: Can we get air in and out unobstructed?

Chediak: There is no question that there is a tremendous challenge to physiologists and scientists to study all these structures. The upper airway is one of the most complex functioning structures of the human body. Look at all the things it has to do. It has to adapt to exercise just like the heart and everything else, but it has to adapt to, for instance, exercise while you speak. It has to adapt to exercise when you stop and drink a glass of water, and you still have to breathe. It has to do all that in a coordinated fashion. Otherwise you aspirate or you choke or you have some horrible clinical consequence. Its various roles, I believe, make it unique as a place for problems in humans and also render it difficult to study.

WATCH FOR A SPECIAL ISSUE OF
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SLEEP-DISORDERED
BREATHING: PART II

MAY 1998

Consequences of Sleep-Disordered Breathing

Kingman P Strohl MD

Introduction
Consequences
  Respiratory Morbidity
  Cardiac & Vascular Morbidity
    Hypertension
    Ischemic Heart Disease
    Myocardial Dysfunction & Arrythmias
    Cerebrovascular Disease
  Neurobehavioral Morbidity
Mortality
Medical Costs & Utilization

Introduction

Patients with sleep-disordered breathing (SDB) may experience a number of potentially adverse physiologic and neurobehavioral problems. The major pathogenic factors in sleep apnea include intermittent hypoxemia and/or hypercapnia, mechanoreceptor activation during obstructed efforts, chemoreflex activation through chronic body and central nervous system excitability, and arousals that result from abnormal breathing. Increased sympathetic nervous system activity potentiates peripheral vascular responses and has chronic effects on the cardiovascular system. In the case of obstructive apneas, large fluctuations in intrathoracic pressure occur and can influence venous return (right ventricular filling), left ventricular filling by interdependence, cardiac output, baroreflexes, and release of volume-regulatory peptides.

The focus of this presentation is on the consequences of SDB as chronic illness in adults. Problems in children are the subject of another presentation. Consequences of SDB are the result of daily exposure to abnormalities in breathing during sleep (apneas, hypopneas, hypoventilation, and respiratory effort-related arousals) and result in long-term morbidity in the neurobehavioral and cardiovascular domains. Data suggest that SDB is associated with increased mortality. Patients with sleep-associated illnesses have poor general health and decreased quality of life as consequences of untreated SDB.

I identified the relevant literature through the review of previous publications from our group[1-5] and others,[5-12] through discussions with colleagues, and by an interactive search using PubMed, an electronic search mode available through the Internet and sponsored by the National Library of Medicine. The reader is directed to recent reviews for discussion and more extensive citation of some of the literature.[1-12] Sections identifying new concepts cite representative primary sources. Terms are defined in Table 1.

Consequences

Respiratory Morbidity

Evidence suggests that SDB can lead to a chronic hypoventilation syndrome both in the presence and absence of comorbidity (eg, obesity, chronic obstructive lung disease, and kyphoscoliosis). Potential mechanisms for this are shown in Figure 1. Obstructive sleep apnea-hypopnea (OSAH) and other

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Table 1. Glossary of Terms

AHI	apnea-hypopnea index	number of apneas and/or hypopneas/hour
BMI	body mass index	weight in kg/body surface area in m^2
CSA	central sleep apnea	apnea resulting from inappropriate respiratory control
COPD	chronic obstructive pulmonary disease	
CPAP	continuous positive airway pressure	
EDS	excessive daytime sleepiness	sleepiness that interferes with wake-time activities
OHS	obesity-hypoventilation syndrome	CO_2 retention in the presence of obesity
OSAH	obstructive sleep apnea hypopnea	apnea and partial obstructions resulting from encroachment of soft tissue in the upper airway
OSAHS	obstructive sleep apnea hypopnea syndrome	signs and symptoms present in OSAH
RDI	respiratory disturbance index	number of respiratory events/hour
SDB	sleep-disordered breathing	all forms of respiratory disturbance (apnea, hypopnea, hypoventilation) and respiratory-effort-related arousals

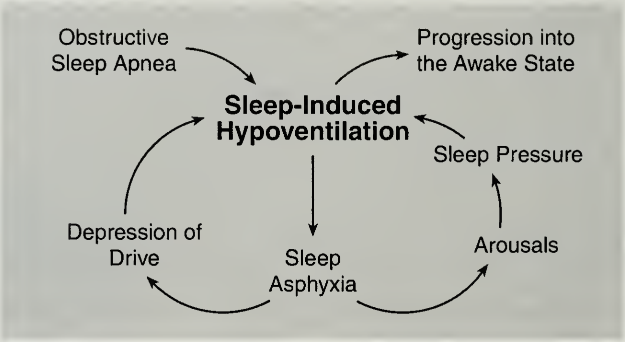

Fig. 1. Ventilatory control abnormalities in sleep-disordered breathing.

forms of SDB lead to hypoventilation and asphyxia. Sleep apnea induces arousals that in turn lead to interruption and fragmentation of sleep, with increased need for sleep or pressure to enter sleep during wakefulness. A secondary pathway to depression of drive during wakefulness is through retention of bicarbonate and metabolic alkalosis from hypoventilation during sleep. Asphyxia during sleep may reduce the production of serotonin and other neuropeptides that in turn influence respiratory drive. Both sleep pressure and sleep-induced hypoventilation may progress to intrude into the waking domain with diurnal respiratory failure. The first evidence for this pathophysiology was demonstrated in patients with respiratory diseases such as neuromuscular illness.[13,14] Management of OSAH either with nasal continuous positive airway pressure (CPAP)[15,16] or with nocturnal ventilation can result in normalization of arterial blood gas values and respiratory drive.[14,17-20]

There is no evidence that SDB by itself is associated with the development of obstructive airways disease; however, there is a co-existence of SDB with COPD ("the overlap syndrome") and symptoms of asthma ("nocturnal wheeze").[21-23] Patients with OSAH are frequently obese, and abnormalities in gas exchange are often considered a consequence of obesity.[3,24] However, OSAH has been associated with biochemical changes that increase insulin levels, which can in turn alter body composition.[3,8] Hypoventilation syndromes in the obese patient are invariably linked to SDB.[4,24]

Cardiac & Vascular Morbidity

Table 2 lists many of the associations between SDB and cardiovascular illness. These have been identified either through epidemiologic studies or large case series. Although the potential for bias exists in all of these studies, it appears clear that SDB increases the risk of developing daytime hypertension and cardiovascular and cerebrovascular illnesses. Compared to data from the National Center for Health Statistics of the general US population, patients with OSAH may have twice as much hypertension, three times as much ischemic heart disease, and four times as much cerebrovascular disease.[2]

Table 2 Cardiovascular Consequences of Obstructive Sleep Apnea

- Confirmed Associations
  - Hypertension
  - Stroke
  - Ischemic heart disease
  - Reduced ventilatory responsiveness
- Suspected Associations
  - Nocturnal angina
  - Right and/or left ventricular hypertrophy
  - Insulin resistance
  - Endothelial cell dysfunction

Hypertension. Data that address the relationship of SDB to hypertension include cross-sectional epidemiologic studies, reports from specialty clinics of hypertension prevalence in OSAH, reports of the prevalence of SDB in hypertensive samples, and studies relating to the severity of SDB to a level of blood pressure.[1,2,7,11,12,18,25] Studies also suggest that sleep apnea patients have reductions in blood pressure and in sympathetic excitation following effective therapy.[26,27] In general, stronger relationships between OSAH and blood pressure have been observed in younger individuals (< 50 years of age). With increasing age, the number and severity of comorbid conditions increase, overwhelming the identification of the specific association.

A number of studies have shown that blood pressure falls following treatment with a tracheostomy or CPAP.[2,5,12] In general, greater blood pressure reductions have been observed in hypertensive patients whose blood pressure profiles show a rise during sleep and in those who are compliant with treatment. Incomplete treatment responses may result from irreversible vascular changes occurring before the diagnosis was made. Heterogeneous responses may be a consequence also of differences among individuals in the effects of OSAH or the operation of risk factors independent of OSAH (eg, obesity, salt sensitivity, and endothelial reactivity).

Studies relating hypertension to SDB can be criticized because of potential confounding effects of obesity, male gender, age, and smoking. However, persistence of significant association after adjustment for these risk factors and demonstration of significant relationships stratified by body mass index (BMI) levels including dose-response relationships between exposure (level of apnea-hypopnea index, or AHI, or respiratory disturbance index, or RDI) and blood pressure partially counter these concerns. The need exists for longitudinal studies and clinical trials to make a definitive assessment of the magnitude of the relationship between SDB and blood pressure, and these are in progress.

Ischemic Heart Disease. The acute hemodynamic stresses associated with apneas are biologically relevant to ischemic heart disease.[2] Much of the evidence to date is based on epidemiologic studies of snoring.[2,5,28,29] History of myocardial infarction has been reported more frequently in snoring compared to nonsnoring people in the community. A clinical case-controlled study of 50 patients with myocardial infarction suggested that symptoms of snoring "every night" increased the odds of myocardial infarction approximately fourfold.[2] A prospective study of more than 4,000 male twins has demonstrated that snoring increases risks of ischemic heart disease (acute myocardial infarction) approximately twofold even when results are adjusted for BMI, hypertension, smoking, and alcohol.[29]

Good evidence suggests that patients with nocturnal angina may have a higher prevalence of OSAH;[30] and, certainly, it would be reasonable to look for evidence of apneas and/or intermittent asphyxia in patients who have known coronary artery disease.

Myocardial Dysfunction & Arrhythmias. Right ventricular and left ventricular hypertrophy occur more commonly in patients with OSAH.[2,5,30,31] Both are attributed to repetitive episodes of hypoxemia, but other factors that occur with obstructed breathing include negative intrathoracic pressures, reduced cardiac output, and increased vascular central volume. The frequency and/or progression of ventricular dysfunction in less severe SDB have been less well described.

Cardiac arrhythmias have been associated with OSAH and central sleep apnea.[5] However, the frequency of serious arrhythmias may be less than previously thought.[2]

Pulmonary hypertension can occur in the absence of structural disease in the lung or the chest wall. It is believed to be related to the chronicity of SDB (ie, the length of time it has existed) but other factors, like obesity, or co-existing disease, like COPD, are believed to be important predisposing conditions.[32]

Cerebrovascular Disease. Data linking SDB and cerebrovascular disease include retrospective studies of patients in whom polysomnography was performed following a stroke, cross-sectional studies of those with a history of snoring, and longitudinal epidemiologic data that have related incident stroke to symptom history. The strongest evidence comes from the Finnish Twin Study[28] that demonstrated that a history of snoring increased combined ischemic heart diseases and stroke approximately twofold. A recent study[33] reported a twofold increase in stroke related to daytime sleepiness and a history of sleeping longer than 8 hours at night; both symptoms are possible markers of SDB.

Neurobehavioral Morbidity

Neurocognitive impairment has been demonstrated best in patients who have high RDI (or AHI) and who also experience moderate-to-severe hypoxemia and sleep fragmentation.[10] Table 3 lists confirmed and suggested neurobehavioral consequences of OSAH. The most commonly recognized feature is excessive daytime sleepiness that is related to the sleep fragmentation and hypoxemia. Figure 2 indicates factors that can act and interact to produce disabling sleepiness. Prior sleep length and circadian changes in sleepiness act independently of OSAH. SDB produces sleep pressures by both sleep interruption and fragmentation and by hypoxemia.[2,5,9,17,34,35]

Table 3. Neurobehavioral Consequences of Obstructive Sleep Apnea

Confirmed Associations
Excessive daytime sleepiness
Attention deficit
Suspected Associations
Learning/memory deficit
General decrease in intellectual ability
Impotence

Some studies have shown a direct correlation between sleepiness and indices of SDB suggesting a dose-response relationship.[9] These studies have limited application because they did not include patients with a full range of respiratory abnormalities, and controls were not comparable in regard to sleep habits, age, BMI, and/or comorbidity. In studies of subjects recruited from the community, subjects with less severe SDB—those that only snore—show little impairment, and mild SDB is not associated with either subjective or objective sleepiness. Alcohol use may confound and/or promote neurobehavioral impairment with SDB.[36]

Other impairments have been identified in general intellectual ability, learning and memory, sustained and focused attention, executive functions, and information-processing efficiency.[9,37] Only a few studies have clearly shown an inverse correlation between some of the measures and indices of SDB, such as low oxygen saturation and/or total apnea time.[2] As has been noted before, these studies have been limited to individuals within several different age and apnea ranges, and there are concerns about controls and potential lack of comparability of controls, given the amount of co-morbidity present in a population recruited from a medical facility.

Sleepiness impacts upon a person's sense of well being.[2,5] Abnormalities in mood and affect have been reported in subjects with heavy snoring as well as in those with OSAH. The major abnormalities are depression, anxiety, and irritability; other areas identified include sexual dysfunction and impotence. Again, these studies may be confounded by co-morbidity. Impairments in OSAH patients presenting to sleep clinics appear to be greater than those recruited from community-based sites. Age is also a confounding factor, and few studies have included women or children in these surveys of neurobehavioral functions.

An important body of literature addresses the effects of established and possible neurobehavioral deficits on the operation of motor vehicles.[2,9,10,35,38] Evidence suggests that the rate of automobile crashes among patients with severe SDB is higher than among the general population. But, even in those with high AHI (> 50), relative risk is modest (twofold) compared to that of cellular telephone (four- to fivefold) and alcohol (10- to 12-fold).[6] There is debate over whether there is a thresh-

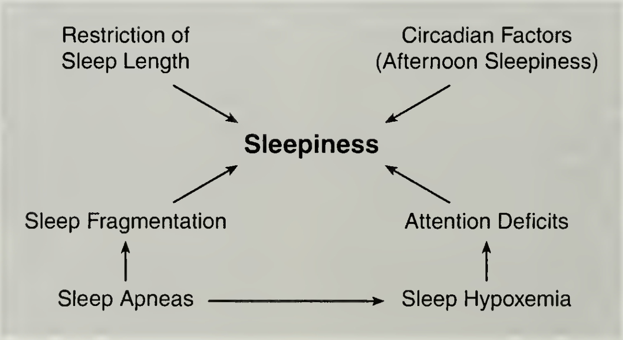

Fig. 2. Factors producing daytime sleepiness in obstructive sleep apnea

old value of AHI above which there is an increased risk of accident or what characteristics of clinical presentation best identifies individuals with SDB who are at highest risk.

The American Thoracic Society[6] has made recommendations that physicians and allied personnel identify the level of sleepiness rather than apneic activity as the risk factor of most concern. One concern is whether health care providers should report patients to the vehicle licensing bureau. Some states at present (1997) require such reporting in a categorical sense (ie, all patients with sleep apnea); however, I have observed that many clinicians take the position that it is the degree of sleepiness rather than OSAH activity that imposes increased risk. For instance, many more fall-asleep accidents occur in younger drivers less than age 30, a segment of the population in which apneic activity may be very low. Sleepiness-related accidents can be attributed more commonly to lifestyle issues and community attitudes toward sleep restriction than to SDB. In absolute numbers, only a minority of accidents are the result of medical conditions. Recognition of the impact of sleepiness on crashes, increasing sleep (sleep extension), and prompt institution of effective therapy are the most effective countermeasures for accidents in patients with SDB. For those without medical disorders, sleep is the most effective countermeasure for sleepiness. Table 4 lists some of the common characteristics of sleepiness-related accidents.

Table 4. Characteristics of Crashes Attributed to Driving while Drowsy

Lone driver
Driving off the road (right or left)
Rural high speed road
Absence of avoidance or corrective action
Poor sleep habits
Early morning hours (2-6 AM)

Mortality

OSA has been postulated to increase mortality through vascular disease, motor vehicle crashes, and work-related accidents. Data to support these observations have been derived from retrospective studies, usually in patients who have presented to sleep laboratories and occasionally from community-based studies of the elderly. The two most commonly cited references demonstrate increased mortality ratios in patients with severe OSA treated with conservative therapy and compare them to patients treated by tracheostomy or CPAP.[39,40] Both of these studies demonstrate excess mortality rates of approximate 6% over 5-8 years, with most deaths attributed to vascular disease. More recent studies have found that all-cause mortality rates are significantly greater in the OSA patient who presents to the sleep laboratory as compared to the general population.[41] After adjustment for BMI, hypertension, and cardiopulmonary disease, AHI predicted all-cause mortality in a dose-dependent fashion; however, the effect was relatively small. After adjusting for underlying cardiopulmonary disorders, vascular-specific mortality was not significantly predicted by level of OSA.

Two of the three community-based mortality studies in sleep apnea focused on elderly subjects. These studies have shown little, if any, effect attributed to SDB. It is possible that older subjects may represent 'survivors' or that older subjects are less susceptible to the pathophysiologic effects of events in SDB. A public health survey identified an increased mortality with symptoms such as sleepiness and sleeping greater than 8 hours, answers generally considered to be surrogate markers for SDB. However, the symptoms are still relatively nonspecific and not tested by measurements of AHI or treatment response.

Table 5 lists some of the factors known or suspected to confound measurements of the impact of SDB on mortality. Age and obesity were discussed earlier. Additional factors include the poor recognition of sleep apnea by medical professionals and health care systems,[5] uncertainty as to what effect the chronicity of OSAH has on cardiovascular risks,[12] and my own observation of no acknowledgment of sleepiness on death records as a reportable proximate cause for death due to accidents.

Table 5. Factors Confounding Mortality Studies of Obstructive Sleep Apnea (OSA)

Age
Confounders: depression, alcohol, obesity
Poor recognition and tracking of OSA by health care systems
No acknowledgment of sleepiness as cause of accidents

These limitations make it difficult to utilize public health records to assess the overall impact and different degrees of SDB on mortality on those of all ages. Nevertheless, the aggregate available data do suggest that mortality effects are likely in individuals with severe levels of OSAH, particularly in those whom OSAH presents in middle life.

Medical Costs & Utilization

The impact of recognition of SDB also resides in the utilization of health care resources and the cost to the patient or society or the health care system for subsequent diagnosis and therapy. An opportunity cost exists in regard to the additional financial burden placed on a person or society by the lost 'opportunity' or absent mechanism for recognizing or treating SDB.[5]

If current estimates of cardiovascular risks for OSAH are correct,[42] untreated OSAH may contribute to 38,000 excess deaths per annum and $3 million (severe disease) to $2 billion (snoring associations) of the health care costs associated with the treatment of vascular diseases may be attributable to untreated OSAH. Behavioral and performance deficits associated with OSAH could carry a considerable hidden cost.[43-45] Reports calculating the sum of morbidity and mortality for motor vehicle crashes and industrial accidents utilize a variety of assumptions to estimate an indirect cost in the hundreds of million to billions of dollars.[44,45] Again, the wide variance in dollar figures illustrates different perceptions of the burden of illness, the need and mode of treatment, and the difficulty in attributing morbidity and mortality specifically to SDB.

No published studies report cost-benefit; however, there are some estimates of the range of potential impact of the illness on health care costs. It is estimated, that 7-18 million people in the U.S. may meet the minimal criterion for OSAH (an AHI > 5); 1.8 to 4 million people may experience more severe illness (AHI > 15 and symptoms of sleepiness).[46] In 1991, dollar estimates related to diagnosis and treatment were between $3 million (for the most severely affected) to $40 billion (for subjects with only heavy snoring). Estimates of improved outcome/unit cost of care suggest a favorable balance in some patients with more severe forms of illness.[47] Whether this degree of benefit would be seen in the wide spectrum of SDB and with all therapeutic options remains open to question. Certainly, prevention efforts would have the greatest impact in regard to cost-benefit. These include educational initiatives to reduce risks, such as sleepiness, weight gain, and upper airways encroachment or increase the likelihood of early recognition of symptomatic illness.

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Discussion

Chediak: How much of the lack of benefit is due to inadequate treatment—either the patient can't tolerate it, the patient doesn't use it (but he says he does), or for whatever reason. What is your estimate?

Strohl: The data that I know are from my own institution, and that's the Veterans Administration Medical Center (in Cleveland). That's a tough group. Those people who have relatively severe disease—that is, are actively sleepy and have nocturnal angina or are on more than two drugs to control their hypertension—those are the ones I want to treat tomorrow. I can get probably 60-70% of those people on some form of noninvasive treatment. They use it at least half the night or half the time. I have no outcome measurements on that group yet. We're trying to collect those data. I think that the reason we have definitions conferences is not to have definitions, but to create entry criteria for outcomes that we would want to track or to do multicenter trials. We, like many other groups, find that in those people who are more severely affected, (those people who have told us they have had car accidents, as an example) about 60-70% of those people say that the treatment for their sleep-disordered breathing/obstructive sleep apnea has changed their behavior and their way of life. These were anecdotal and relative to our center. To generalize across centers—in the literature those that report the data, regardless of their definition of a hypopnea, are consistently showing benefits. In Europe with uvulopalatolaryngoplasty or with CPAP, reports suggest decreasing symptoms of sleepiness, car accidents, and blood pressure. These results are fairly consistent, even if they don't use the same definition. We're talking about the same sort of thing. Now, how can we define it better?

Stoller: Kingman, you have pointed out the risk of motor vehicle accidents. Let me ask a public health question in that regard. When we identify drunk drivers, we generally react by insisting on license suspension or corrective action—AA [Alcoholics Anonymous] kind of instruction. Given the under-recognition of sleep apnea, and the fact that there may be accidents attributable to sleep apnea in the severe range that you've identified, should we be campaigning for screening or for case finding? Should we be campaigning for a public policy to identify accidents with the high risk characteristics you've identified (where cellular phones and alcohol are not implicated) and mandate sleep testing in those individuals? Is this an active issue or should it be if it isn't?

Strohl: I've been on several panels and have thought about this. The report of the American Thoracic Society, I think, lays out in an academic fashion some of the arguments against having mandatory referral and pushes more for an educational mandate to three groups—first, the primary care physician, to identify sleepy patients; second the licensing bureaus, to understand and have a common policy for trying to do this; and third, for the patients to become aware that this is an issue. The other group around the table is society itself who will judge what the criteria will be. As a physician, I do not license drivers. I am not mandated by the state to give a license. I cannot charge a fee to give them a license. I can't say that they can drive a car. I don't have training for that. I can say whether a person is sleepy or has been treated. The test in the laboratory does not necessarily predict how patients will behave, and that has been shown for Alzheimer's, for epilepsy, and for other disorders. I think that this is an issue for public debate. Usually when physicians, as they did in Canada, get involved and have mandatory reporting, then five years later they back off, because it keeps patients from coming in and getting therapy. So, that's one side. My mother began driving an automobile in 1924 when her parents said "Now you can drive." She was 14. She didn't have seat belts. There were no lights; there were no stop lights; there were no alcohol laws. There was nothing. And no physicians necessarily got up and said "Gee, we have to have all these things." Society will help us define the threshold that they want. When the degree of sleepiness that produces automobile accidents becomes unacceptable to society, then we will have an informed debate. But there's no test at the time of an accident, unlike alcohol, that you can use to define sleepiness.

Erman: This has obviously been a source of controversy in the field. We had a program several years ago at Scripps Clinic that considered sleep and public policy issues. At that meeting, we had a representative of the Department of Motor Vehicles (DMV) in California. Physicians do have a responsibility in California to report patients with lapses of consciousness to the county health authorities. You aren't reporting to the DMV, but the county health authorities then report them to the DMV, which will then call in patients with defined lapses of consciousness to determine whether their licenses should be pulled. Somewhat to our surprise (this was 3-4 years ago), it turned out that without any specific input from the medical field the DMV in California had already characterized sleep apnea as a lapse of consciousness. No question. This was in their mind a reportable condition, and physicians were obligated to report this to the county health director as an issue of public safety. This has not, obviously, been followed fully by physicians, but it is a matter of law and public record. Second, interestingly, the FAA (Federal Aviation Administration) has decided that in pilots with private pilot licenses, not commercial pilots, that sleep apnea *is* a medical problem. Pilots who are treated with CPAP and who have a normal Maintenance of Wakefulness Test (MWT), which is the indicator they elected to use, can continue to fly and maintain their licenses. So, decisions about sleepiness and sleep disorders are being applied in a haphazard fashion with regard to issues of public policy and public safety.

Strohl: Your point is well taken, and you have to know your state's law. California is one, and I believe that Arizona, New Mexico, and Florida may also have similar sorts of laws. There remain 46 other states that may or may not have them, and at the time that this is published, that could change one way or the other. So, it's not worthwhile to have a list. The other side is that ignorance of the law is not a defense, so your state societies need to keep on top of this and be able to communicate these issues.

Woodson: One of the challenges, I think, to the field is to develop outcome measures other than things like MWTs to be able to measure these things because right now it's very difficult to assess sleepiness in the office. We certainly recognize it in the severe patient, but without good outcomes, it's very difficult to establish policy or to make clinical decisions.

Brooks: You mentioned the patient who might be so severe that you would treat him without a study. This is an area of controversy in pediatrics. Some people suggest that a patient with severe disease should be hospitalized or put on CPAP prior to surgery. There are no data that I'm aware of that tell us about the relationship between the severity of disease and outcome in kids. How do you decide that a patient is so sick that you don't need a study, you're going to treat him immediately?

Strohl: The concept is to use a threshold approach to decision making.[1-3] For any test there are those two possibilities—a test threshold and a treat threshold. I've been in the intensive care unit and rung the alarm bells for obstructive sleep apnea (those in hypercapnic respiratory failure who are falling asleep and are unable to cooperate with noninvasive therapy). I wouldn't wait to schedule a sleep study; I would treat before testing. I would do sleep studies at a later time once the patient is stabilized. On the other hand, there are people whom I do not test, at least immediately. I think that we need to articulate what we think those thresholds are, and, perhaps, each center should agree on what those thresholds are. Then, say, once I have done this, follow those patients and say what their outcome is. If certain centers believe that severe OSA is anyone who has an AHI > 15, is sleepy, and has an oxygen saturation $< 85\%$, then one year later, I want to see how many of the patients whom they treated are now improved. I would suspect that those groups would have a lot of people who probably aren't using their therapy and probably have consumed a lot of time and effort. On the other hand, if you had a criterion that those with severe OSA were excessively sleepy and had AHI > 40, I think you would find the group that shows a higher compliance with nasal CPAP, for instance, and so I think those are the thresholds we need to articulate for outcome studies.

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Pierson: Do you think there are circumstances in which an evaluation for sleep-disordered breathing is not clinically valuable? I'm thinking specifically of the scenario you were just visiting—that is, the patient we first become aware of when they're in the ICU or acutely ill in the hospital, when we're summoned to evaluate the patient for sleep disorder at a time when they're medically very sick.

Strohl: The answer to that is Certainly. I think it goes both ways. I think there are certain people in whom you would know from prediction profiles that they have some apneas, but you may not test them because you may not treat them in a way that would require a laboratory test.

Pierson: I don't mean never to work them up or treat them, but …

Strohl: I agree; you would wait. If sleep apnea-hypopnea syndrome is as common as diabetes or as asthma, we have to think of recognition and a variety of diagnostic strategies, many of which are valid.

Pierson: I think that the commonest circumstance in which diagnostic workup for a sleep disorder is requested in our university system is in that setting—when the patient is medically very ill and unstable.

Strohl: I think that sleep apnea occurs in the home, should be treated in the home, should be diagnosed in the home, and that sleepiness can be a product of hospitalization and illness; so I usually like to stabilize people and preferably test them as an outpatient. I think there's a misperception in the medical community that a patient being treated for congestive heart failure with diuretics and an I.V. is a perfect candidate for a sleep study. I don't think that's true—whatever type of sleep study.

Minkley: I think it is important to point out the need for some definitions of terminology. When we talk about evaluation of the sleepy patient, what do we mean by evaluation? Evaluation could mean a diagnostic test, full polysomnography. Evaluation could mean an investigation of patient's history, symptoms, by an adequately trained professional like you, who will come to a conclusion and institute treatment. I know that in sleep-disorder centers across the country this becomes an issue when a call is received asking for a sleep evaluation and the need exists to clarify—What do you mean? Do you want a sleep study? What kind? Do you have a patient who needs to be seen by a sleep specialist? Most frequently the response to that is I don't know.

Strohl: Our response to that is first a history and physical. In the ideal world, we'd be able to have guidelines for that primary care physician to have at least a pretest probability in his or her own mind or be able to articulate what the degree of illness is in that patient. Perhaps the role of the sleep center is to help the primary care practitioner articulate that even before the patient comes to the center. That would be in an HMO and preventive medicine mode.

Minkley: Would that not actually be the case in the ICU scenario we were talking about, because the patient described may not need to be evaluated by some diagnostic tool or instrumentation. In fact, the trained clinician may be the tool.

Strohl: The ICU example is to really articulate that there are thresholds that we currently use and have used for years, even before we knew the term 'sleep apnea.' Twenty years ago, we intubated these patients, and they were better in 3 days so we extubated them and they felt great. Six months later they came back in. But in this group we now initiate CPAP without a 'sleep study.' I think the problem is the mild-to-moderate group in which we have to articulate what therapy and how do we want to do this? I think we begin with a history and physical.

Barnes: I'd like to second that. My experience was with a Pickwickian, a coworker, who fell asleep during meetings. He would fall asleep and be able to pick up the discussion immediately upon waking. We know more in 1997, but how easy is it for someone to have a sleep study done? Is it easily reimbursed by health insurance? Is it time-consuming? What kind of motivating factors could I have used or would you use, to get someone to take the time to do that?

Strohl: You can 'drive a horse to water.' If the light bulb can click in the patient or his family that they have an illness and if we can state that we think that this is amenable to treatment—that requires an educational rather than mandatory process. Patients can be referred for assessment either because they raise their hand to say they think they have it or because the light bulb comes on in the physician's head. We don't know enough about those strategies. One of the first patients I had was referred in 1978 because of a *Time* magazine article. I think we now have better patient-oriented material. The National Center on Sleep Disorders Research (a division of the NHLBI) has patient-oriented material, physician-oriented material that is available upon request at a very nominal fee—100 of these things for $10.* Materials from the National Sleep Foundation, the American Sleep Apnea Association, getting other patients to call them who have been successfully treated, using patient care groups—all can be effective in those health systems in which the diagnosis and treatment of sleep apnea is regarded with some skepticism. Some suggest that sleep apnea is the cardiac murmur syndrome of the 90s, that is, that everyone needs a sleep study because they snore. We have to make sure who needs a study—when and where and why—again to articulate the level of illness that this patient has, the fact that there is therapy that can be provided, and that there is treatment that can change quality of life. It's not a guarantee, but this is a process that we have to articulate, verify, and refine. I think you have to educate as to what we know now, and what you try to do in the absence of better information, what is not known, and what needs to be known. Certainly it's not just to physicians; it's also to respiratory therapists, other technicians, other members of the health care team, including nurses.

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*For further information call the National Center at 1-301-435-0199 or write: Bethesda MD 20892.

Mathews: We do teaching rounds in the ICUs in our facility with our students and over the last 6 months, we've seen an increasing recognition of sleep disorders. It's kind of becoming the disease of the half-year. The nursing journals are now writing about sleep apnea. It seems every time we go up into the ICU, be it medical, surgical, or transplant, the nurses are calling up and saying "This guy's not sleeping well. Does he need a sleep study?" "Well, when was the last time you woke him up?" "We have him on q-hour vital signs." We're coming in and interrupting his sleep, fragmenting his sleep, continuously, yet it is being perceived by the caregivers that these people have a sleep disorder. Well, the sleep disorder, to my mind, is the interruptions that we impose on this person's sleep. I think what we need to do is consult the caregivers in the ICUs to figure out ways of monitoring patients that do not interrupt their sleep. If you have been an ICU patient, you know that there's no way to get any sleep. Every time an alarm goes off, you *know* it's your alarm. You're wide awake, and your EKG starts spiking because "That was me," even if it was the patient down the hall. So, one of the messages that I think needs to come out of this conference is that we need to be—as you've been saying—very selective about whom we do and don't treat; but we also need to be selective in the types of education we give, maybe to the nonphysician, nonrespiratory care caregivers.

Chediak: The notion that improving the quality of life of a human being by therapeutic intervention is valuable and is to me key to your allegory about when to treat and when not to treat. I was just thinking. When does improving the quality of life also include increasing the quality of life of spouse and others who have to put up with the noise at night? Is that something we should look at or simply ignore and look only at the health consequences? Because in the end, if your spouse is sleeping in a separate bedroom and that produces a certain amount of emotional strife or even a certain amount of sleep disruption because she or he keeps waking you or you have to leave the bedroom, and you have sleep fragmentation, then you're sleepy—not just because you snore and maybe have mild-to-moderate sleep apnea, which is inconsequential to your health, but you have sleepiness because you're spouse keeps waking you up and you keep having to move from the bed to a couch and vice versa. Apart from that: Wouldn't it be nice if we had a serum marker of sleepiness, like a cytokine S level, that we could just draw in the field when you are in an accident and there it is. It's high, so you knew you were sleepy when you were in this accident. It's a challenge to us to find either some somnagenic cytokine or neurotransmitter that we can correlate with sleepiness, so that we can make the measurement of sleepiness a little bit simpler for all of us.

Strohl: Alex, cholesterol screening is now down the tubes.

Chediak: We can do CSF adenosine levels. The only problem is getting that lumbar puncture.

Strohl: But you know, we find that everybody's sleepy. Thirty percent of the population is sleepy because of sleep restriction. Let's go back to the other thing. John Shepard recently identified the "spousal-arousal syndrome," spouses of sleep apnea patients. That's a great term for a problem of sleep habits and environment. You've identified an important omission in what I talked about when we articulate our goals of therapy with a patient and the health care system—the consequences to the family. I think if we articulate that this is a person in whom we want to treat loud snoring, then this is a person in whom we want to treat *this*. We now have articulated that outcome to the patient. We've articulated, perhaps, that a strategy to be able to attain that might be different for a patient with excessive sleepiness than for loud snoring. We're now starting to do this because snoring is too common—50% of the men snore and 30% of the women snore. You then begin to discuss treatments ranging from external nasal dilators and mouth guards from the dental profession to reconstructive surgery of the face and jaw that have an enormous up-front cost. It really depends on whether we articulate the goal of therapy for that patient and whether we can show what the outcome is. I think that's really where we're at. I think it's a clinical era, rather than a polysomnographic era.

Minkley: I wonder if you have a sense for how often the other goals of therapy are addressed with patients—that is the neurophysiologic consequences of sleepiness—performance deficits, personality changes, in males impotence, all of those issues, which may or may not get addressed at all. I believe the general public is used to coming to a physician with something that's broken and asking to have it fixed, or "I have a cold" or whatever, but in the sleep-disordered patients, more frequently, they're being brought by someone to fix something they don't believe they have a problem with.

Strohl: I agree with that sentiment because of the variety of people who walk in for those various reasons. I think the evidence right now suggests attention deficits that people may or may not realize, and sleepiness, which they do realize. But what do I want to treat tomorrow? As you go down this ladder from severe to moderate to mild, a lot of the confounding variables of age, comorbidity, start to impinge symptoms—impotence and memory and intellectual ability, and some may be restored and some not. I think we have to be careful about saying what we can fix and what we can't. We need evidence-based decisions. For instance, I don't use CPAP to treat hypertension or impotence as a first-line therapy; there are established treatments for these problems that are known to improve morbidity and mortality. So, we have to work with other people and articulate where we think our role is going to be in that provider sense. We can't promise more than what we can deliver.

Erman: I think there are fairly good data with regard to other behavioral areas, intellectual function, and mood. There are a number of studies going back many years[1-3] that show improvement in mood and intellectual function, post- versus pretreatment. The problem with these studies is that they are all post hoc. They are not controlled. You don't have prospective data. But I think there are adequate data supporting the notion that this is also a relatively well-defined benefit.

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Strohl: It's a very soft literature. It may show ascertainment bias by whom they studied. There are referral biases. In regard to interaction with psychiatry and mental illness, in particular, the clearest example that I've seen recently is the fact that the presence of a high depression score by Beck's inventory should not preclude a sleep study. I think that's an important clinical point. The other is that in some of these studies, the oddity occurs in the severely affected person that the sleepiness really dominates many results of neuropsychologic tests. In fact, subjects could get neuropsychologically worse in some tests with treatment because they become more alert. It's a very complex but interesting area. Again, it is important to try to articulate where our role is going to be with our psychiatric colleagues and their research.

SEE PAGE 342

For a listing of sleep organizations in America

Clinical Diagnosis of Sleep Apnea

Barbara A Phillips MD MSPH FCCP

Introduction
Medical History
  Sleepiness
  Snoring
  Apneas
  Cognitive Dysfunction
  Impotence
  Headache
  Other Symptoms
Physical Findings
  Obesity
  Hypertension
  Nasopharyngeal Narrowing
  Other Physical Findings
Anthropomorphic Variables
  Gender
  Age
Clinical Prediction Rules
In Conclusion

Introduction

Clinical findings in patients who have sleep-disordered breathing (SDB) are currently the topic of intense investigation. It is likely in the future that the history and physical examination will be used as tools to predict the presence or absence of sleep-disordered breathing and obviate the need for polysomnography. However, the clinical diagnosis of obstructive sleep apnea (OSA) currently depends on objective measurement of sleep and breathing variables in a laboratory. Much has been written about findings on medical history and physical examination in patients who have this condition. Unfortunately the issue of what symptoms predict the presence of sleep apnea is clouded by the fact that the definition of sleep apnea is not precise. Screening of population samples has yielded a high prevalence of patients who meet the standard criteria for the diagnosis of sleep apnea.[1,2] However, many of the patients identified as having 'sleep apnea' in asymptomatic populations are probably not sick at all. This undoubtedly contributes to the dearth of evidence that sleep apnea causes harm and that treatment is beneficial.[3]

For purposes of this discussion, I focus on patients with clinically important OSA. Specific findings on history taking and physical examination that correlate with or predict OSA are included in this discussion.

Medical History

The symptoms and signs that are classically associated with sleep apnea are outlined in Table 1.[4] The diagnostic criteria for OSA are outlined in Table 2.[5] Certain features are common to these and other descriptions of patients with OSA;

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Table 1. Patients at Risk for Sleep Apnea*

Symptoms
- Chronic, loud snoring
- Gasping or choking episodes during sleep
- Excessive daytime sleepiness (especially drowsy driving)
- Automobile or work-related accidents due to fatigue
- Personality changes or cognitive difficulties related to fatigue

Signs
- Obesity, especially nuchal obesity (neck size > 17 in. in men, > 16 in. in women)
- Systemic hypertension
- Nasopharyngeal narrowing
- Pulmonary hypertension (rarely)
- Cor pulmonale (rarely)

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Table 2. Diagnostic Criteria: Obstructive Sleep Apnea Syndrome (780.53-0)*

A. The patient has a complaint of excessive sleepiness or insomnia. Occasionally the patient may be unaware of clinical features that are observed by others.

B. Frequent episodes of obstructed breathing during sleep.

C. Associated features include:
  1. Loud snoring
  2. Morning headaches
  3. A dry mouth upon awakening
  4. Chest retraction during sleep in young children

D. Polysomnographic monitoring demonstrates:
  1. More than 5 obstructive apneas, greater than 10 seconds in duration, per hour of sleep and one more of the following:
  2. Frequent arousals from sleep associated with the apneas;
  3. Bradycardia or tachycardia
  4. Arterial oxygen desaturation in association with the apneic episodes; with or without
  5. A mean sleep latency test (MSLT) that demonstrates a mean sleep latency of less than 10 minutes.

E. Can be associated with other medical disorders, eg, tonsillar enlargement.

F. Other sleep disorders can be present, eg, periodic limb movement disorder or narcolepsy.

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sleepiness, snoring, personality changes, headaches, and witnessed apneas are the classically reiterated clinical symptoms of this condition.

Sleepiness

The use of excessive daytime sleepiness as a clinical symptom presents several problems. The first is the definition of sleepiness itself. Many writers and government publications equate sleepiness with fatigue. These are not the same thing. Sleepiness is the urge or tendency to fall asleep. Fatigue is tiredness, which may not predispose to sleep. Sleepiness results from sleep loss or sleep pathology, and fatigue results from exercise or other causes. It is not surprising, then, that studies evaluating the usefulness of the clinical symptom of sleepiness have found it to be neither sensitive nor specific for OSA.[6-8] Falling asleep while driving does appear to have predictive value in patients suspected of having sleep apnea.[9] Despite this lack of proven utility of self-reported sleepiness in establishing diagnosis in OSA, sleepiness is still considered a hallmark of sleep-disordered breathing.

A tool commonly used to assess sleepiness in clinical populations is the Epworth Sleepiness Scale.[10] In general, patients with sleep apnea score higher on this instrument than patients without sleep pathology.

Snoring

A plethora of studies report the prevalence and sequelae of snoring. As many as 50% of men and 25% of women snore,[11] but only 4% of men and 2% of women have sleep apnea.[12] Thus, snoring is clearly not a specific indicator of the presence of sleep apnea. In clinical studies of patients with sleep apnea, snoring is virtually always present.[9] The usefulness of the symptom of snoring as a discriminator probably depends on how the question is asked; questionnaires that are typically used in sleep laboratories query patients and their bed partners about "loud, disruptive snoring." In one study,[9] the sensitivity of snoring intensity in predicting apnea activity increased with the intensity of snoring.

Apneas

The presence of witnessed apneas is specific for the presence of OSA.[6,9,13-15] This finding appears to be consistent across studies utilizing this marker. Many patients who have sleep apnea will be unaccompanied by a bed partner, however, and their evaluation may not be able to include this important finding.

Cognitive Dysfunction

Patients with sleep-disordered breathing commonly exhibit impaired daytime cognitive function.[16] Cheshire and co-workers[16] demonstrated that the degree of cognitive impairment is correlated with severity of SDB, magnitude of oxygen desaturation, and degree of sleep disturbance. My colleagues and I[17] compared oxygen administration to continuous positive airway pressure (CPAP) in treatment of patients with moderate sleep apnea. Patients treated with either therapy showed improvement in cognitive function. It appears that both hypoxemia and sleep disturbance contribute to daytime cognitive dysfunction in patients with OSA. Despite the robustness of this finding, it is often difficult to elicit a history of impaired cognition from either patients or their partners. Nevertheless, individuals who volunteer history of disturbed daytime performance should be taken seriously.

Impotence

Impotence is reported in clinical OSA populations with variable robustness.[6,18] Because the symptom applies to only half of the population and is difficult to elicit from some patients, it is usually not clinically useful in discriminating between those patients with sleep apnea and those without.

Headache

Headache is known to be a part of the sleep apnea syndrome and in reports[19-22]is typically characterized as morning headache. In a careful study of patients with OSA and those without, Ulfberg and colleagues[19] found a prevalence of 5% of morning headache in the general population, compared with 18% in patients with OSA.

Other Symptoms

Other symptoms that are frequently reported to be present in patients with OSA include nasal obstruction,[23] restless sleep,[18] and recent weight gain.[15]

Physical Findings

Obesity

Obesity is the single most important risk factor for OSA, and its presence is highly predictive of the disorder. Central obesity, particularly large neck circumference, is particularly likely to predispose the patient to the development of SDB.[24] Men who have necks bigger than 17 in. and women who have necks bigger than 16 in. are at much higher risk for OSA.[25] In fact, most sleep apnea patients are obese and weight loss is beneficial for them.[24]

As a predictor of the presence of OSA, body mass index (BMI) has been most frequently evaluated in the literature. Viner found that patients with OSA had an average BMI of 30 ± 6 kg/m^2 as compared with 27 ± 4 for their non-apneic counterparts.[18] They found that only age, BMI, male gender, and snoring were predictors of sleep apnea in their sample.

Hypertension

Systemic hypertension is more common is patients with OSA than it is in the general population. Conversely, there is a higher prevalence of OSA in hypertensive subjects than in the general population. Nevertheless, a careful review of the health effects of OSA in 1991 concluded that the evidence of a causal association was still lacking because of the confounding influence of obesity.[26] Further studies[27-31] have found no associations between sleep apnea and blood pressure, but others have found statistically significant association with morning elevations of blood pressure.[32-34] Although many studies have found an association between sleep apnea and daytime systemic hypertension, most of these have not adjusted for the effects of cigarette smoking, alcohol, or antihypertensive medications.[3] Furthermore, at least two studies have evaluated hypertension as an outcome measure of CPAP treatment and found no benefit.[17,35] Thus, although conventional clinical wisdom relates hypertension to SDB, the evidence is still somewhat shaky, primarily because of failure to control for confounders such as obesity and cigarette smoking.

Nasopharyngeal Narrowing

Although results of examination of the posterior of the oropharynx are frequently reported in the literature, they are subjective findings. Surprisingly, however, the results of the pharyngeal examination do predict the presence of sleep apnea.[6,18] In particular, the presence of tonsils or an enlarged uvula, as noted by a seasoned clinician, are important predictors of the presence of OSA.

Other Physical Findings

Although pulmonary hypertension and cor pulmonale have been reported to be associated with SDB, their relationship to sleep apnea is actually not well established.[3]

Anthropomorphic Variables

Gender

Unquestionably, male gender poses a risk for OSA across all boundaries of age, ethnicity, and weight.[4-12] The male-to-female ratio, however, is probably 2:1, rather than a much higher ratio as has been previously reported.[12] Gender is an important predictor of the presence of sleep apnea in several prediction models.

Age

The peak prevalence of clinically significant OSA is probably in the 40s or 50s.[1,2] Although the number of apneic events in non-clinical populations appears to increase with age,[36] this may not correlate at all with pathology.[37,38]

Clinical Prediction Rules

Because of the expense and inconvenience of overnight polysomnography, several investigators have attempted to construct models, questionnaires, and algorithms that can predict the presence of OSA without polysomnography.[7,9,14,15,37] Although these models are often superior to physician impression,[14] they lack sensitivity and specificity and appear unlikely to substantially impact the ability to diagnose OSA outside the laboratory or to reduce cost.[39]

In Conclusion

The historical findings that are predictive of sleep apnea include witnessed apneas, daytime sleepiness, and heavy snoring. Physical findings that have been linked to sleep apnea include obesity and hypertension. Several clinical prediction rules and algorithms have been developed using combinations of these parameters in order to predict the presence or absence of sleep-disordered breathing. Unfortunately, this science is still in its infancy and lacks enough sensitivity and specificity to replace clinical testing of patients suspected of obstructive sleep apnea.

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Discussion

Phillips: It is good, I think, to point out the really famous people who almost certainly had sleep apnea. John Candy, who did die in his sleep, is the example that I often use when talking to lay audiences. Oh, come on Tucker (Woodson) I know you have something to say about all this.

Woodson: I think it is important to understand that the Epworth Sleepiness Scale[1] was developed as an evaluative measure to follow population treatment effects. It was not developed as a discriminative measure to distinguish those people who have sleepiness from those who do not. It is my experience that the Epworth scale is being used as a discriminative tool and not as an evaluative tool. For example, insurance companies ask "What was their Epworth score?" If a patient's Epworth score is less than x, y, or z, then they won't cover a sleep study. Usually, when an Epworth is positive it means something, but a low Epworth does not rule out sleepiness.

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Phillips: I absolutely agree. And it hasn't been found to be useful in studies looking at clinical prediction rules.[1-3] Sleepiness as a symptom is worthless. Now, car wrecks due to sleepiness are meaningful, and I think we need to iterate that, because I, too, have patients who are referred for sleep study whose insurance company rejects payment for sleep study because no one documented that the patient was sleepy in their pretesting evaluation. Who cares? Some of my patients are so sleepy that they don't know they're sleepy. Of course, a significant minority of people who have sleep apnea actually complain of insomnia. Thank you for making that point. We all need to shout it from the rooftops.

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Erman: I just want to strongly support the recognition that sleep apnea often presents with insomnia complaints—probably more often complaints of nonrestorative sleep than of nocturnal awakening. The assumption is made too often that because the patient's primary or presenting complaint is not one of daytime sleepiness, they don't have sleep apnea. I think it's a common finding and one that should really drive much more investigation into these symptoms than occurs at present.

Strohl: The bed partner issue is certainly there, but there are few studies that have tried to determine whether it really makes sense to try to get information independently from the bed partner of the patient. In some ways it depends on how you ask the question. If you ask the question "Do you snore?" or "Do you stop breathing at night?" the answer is "I don't know. I'm asleep." But if you ask "Has anyone ever told you that you stop breathing at night? Do you snore loudly enough to be heard in the next room?" And then go on to say "Well, use any source of information, such as camping trips, business trips." You get an answer, especially for more severe illness. So that's one extreme. They usually have taught that. That's at least been my experience, and I think the Kump et al study[1] showed that it probably wasn't worthwhile to do 2 questionnaires, one to the spouse and one to the patient. Is that your thinking?

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Phillips: I absolutely agree. I think it was Kump who showed that the predictive value of snoring as a symptom increased with the intensity of the modifier used to describe it. But yet, a lot of people do say "I was on a camping trip last week, and the guys made me sleep out in the rain." Or whatever. Yeah, that's right. People who are single often do know that they have trouble with their breathing during sleep. In fact, people who have trouble with their breathing during sleep may have an increased risk of being single.

Strohl: In either asking a patient or having a patient fill out a questionnaire with help, the category of "Don't know" becomes important and should be on most questionnaires or screening because "don't know" really means that. It should not dissuade you one way or the other, but it's an important variable. The other side that's interesting is the different presentations in women and men, at least in the community-based studies. For the same amount of apneic activity, women have more complaints of fatigue, and men have more complaints of snoring—whatever that language means, at least in the U.S. studies. It may be culturally sensitive as well. We probably need to know more about what people think about with fatigue and be clear about their meaning and how we define it as doctors or scientists.

Phillips: I would like to say, women, in the aggregate—leaving the issue of sleep apnea aside—are more likely to complain of fatigue than their male counterparts, although self-reported fatigue in men has increased over this century, as you probably know.

Epstein: The question I am asked most often by primary care physicians is "Who do I send for a sleep study? How do I tell?" I think our experience is a bit skewed. By the time patients get to us in our sleep clinics, the primary physicians have already sorted out the people with the most severe disease. And the people who have the mildest disease are the ones all of our measures are least sensitive in detecting. How would you answer that question about deciding who to send for further testing?

Phillips: Yes. I am asked that question a lot, too. I think one way of viewing it is to ask which people, if diagnosed with sleep apnea, would benefit from treatment? So, if the patient doesn't have sequelae of sleep apnea that you would expect to get better were he treated, then it may not make sense to study that patient in a laboratory. So, if the patient and the people who know that patient don't complain of cognitive dysfunction or sleepiness, if that patient doesn't have witnessed apneas that are disturbing the bed partner, if that patient isn't hypertensive or doesn't have cardiopulmonary disease, then, perhaps, that patient wouldn't benefit from having the disease detected, even if it were there. But to look at the clinical prediction rules, I tell them I study everybody who has a neck size over 17 in men, 16 in women, who has witnessed apneas, hypertension, and/or automobile accidents. That's the way I do it. Now, there are clinical predictions, and I think Flemmons's is the best.[1] There are clinical prediction rules that can be used that give you a high pretest probability that your study is going to be positive. The problem is that they leave out patients who would also have a positive study, who might benefit from treatment. I'd be interested in how others answer this question. Yeah, Lee. Are your patients all babies?

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Brooks: They're not all babies, and I do see adults, too. And I answer the question differently if the primary doc calls me on the phone and says "What should I do?" compared to a self-referred patient. I think there's been a different level of screening there. If the patient calls me up and says "I snore" I want to see something else before they come into the lab. That could be daytime sleepiness, difficulty concentrating, or witnessed apneas. Snoring plus something else. Usually by the time they find their way to see me, they're going to get a study because their physician has said this is different than all the other 40% of men that I see who snore. Even in the absence of other symptoms, I'm likely to study that person because of the level of screening that's been done by the primary physician. I think, also, that we can't tell who we're going to treat because we may be able to discern the level of cognitive function or sleepiness, but we can't tell the desaturations. So, I think it would be hard to say "I think this is severe enough that I would treat it anyway, and, therefore, I'm not going to study it because I don't know what their oxygenation is doing."

Phillips: I think this is a critical issue that Larry (Epstein) has brought up because where we set the bar determines a lot of things—from, perhaps, how much death there is on our highways to how many billions of dollars are spent on CPAP and ENT surgery per annum. I don't really claim to have the right answer to this. I know that every week I diagnose sleep apnea in patients who cannot or will not accept treatment because the treatment seems worse than the disease to them. I don't know how to deal with that. Kingman (Strohl), I know you have a thought about this.

Strohl: I have identified two other groups that I might have a primary care practitioner think about, and there is some evidence, but not a lot. The first group would be those who have nocturnal angina. I based that on biological plausibility and some studies from England[1,2] that have shown that in those presenting with angina, those who have a nocturnal component to it, have a high likelihood of having sleep-disordered breathing. I think that may not need a lot of study, but that's a risk group. The other would be patients in whom blood pressure is not controlled by two or more medications. Again, these are people who our primary care practitioner would run across and be having trouble with. I'd like for them to think about sleep apnea even if they do refer the first type of patient to a cardiologist and the second to a hypertension specialist. I'd like those cardiologists and hypertension people to think about sleep apnea. Is that reasonable?

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Phillips: That seems very reasonable to me.

Atwood: Kingman, have there been studies to look at the effect of CPAP on these poorly controlled hypertensives or is this where biologic plausibility and intuition come into play?

Strohl: In the groups in which sleep apnea is looked for—hypertensive groups —the more medications you're on, the more likely you are to have sleep apnea. That's in some of that literature.[1-4] There are some suggestions of a reduction in blood pressure or a reduction in the number of medications needed to control blood pressure with treatment of sleep apnea.[5] I've not seen anything that looks directly at that. The studies on nocturnal angina are those from the Public Health Service in Britain[6] and are just really an odds ratio of likelihood. These reports don't follow a treatment or outcome effect. So, I think that when there are indications other than just sleepiness that you might then look at those people a little more carefully.

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Phillips: I think the Sleep Heart Health Study may help us out with this a little bit—many of us are hoping that it will.

Brooks: Kingman, were you suggesting that a patient with nocturnal angina or uncontrolled blood pressure should have a polysomnogram or just that you'd think about a study?

Strohl: There are a finite number of reasons for having nocturnal angina and uncontrolled hypertension, and sleep apnea is one of those reasons. The evidence suggests that it is a very strong predictor and one that we haven't previously appreciated. There should be structured questioning, not just a test response.

Brooks: The group that we thought would fit into that category were patients with sickle cell anemia who were having frequent crises. Our idea was "Well we don't see how these guys who are sick are different from these guys who aren't," and we hypothesized that nocturnal desaturation and, perhaps, OSA was the difference. But when we studied it, we didn't find that to be true.[1] Although there were patients who had OSA with the sickle cell disease, when we separated kids who were in frequent crisis from those who were not, there was no difference in the polysomnograms between them, nor in their pulmonary function.

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Mathews: As a non-sleep professional, can I get two definitions from the group. First, what is the normal amount of sleep that one should see in a normal individual?

Phillips: 8.3 hours a night!

Mathews: Uh-huh. OK, that's one I've heard—about 8 hours. I've also heard anything over 6.

Phillips: Or how about 5-10 hours?

Mathews: 5-10? OK.

Phillips: Either way.

Mathews: It's nice to have something to shoot at. The second question is probably even more telling. What does excessive sleepiness mean?

Phillips: Excessive daytime sleepiness?

Mathews: Yes. What does that mean?

Phillips: It is a garbage term, isn't it? And it actually presupposes that there's some normal amount of daytime sleepiness. I have no idea. But it is a symptom that when a patient or somebody who knows a patient complains of it means that person seems less alert in the same circumstances than counterparts do, but it is highly subjective. For example, I have patients tell me "I doze off at my computer every afternoon after lunch. That's normal, isn't it?" Well, I don't think so. And I have patients brought in by their wives who are concerned because they're falling asleep in church and that is normal, as far as I know. So, you're right.

Mathews: So, it's kind of like an elephant. You'll know it when you see it?

Phillips: Or pornography—either one. I think it's equally as slippery a distinction to make. Now if somebody else feels that he or she has a really good definition of excessive as opposed to normal daytime sleepiness, I would love to hear it.

McIntyre: You alluded to hereditary traits. Again, I'll have to plead ignorance because I'm not in sleep, but are there data to support genetics or has that been looked at?

Phillips: I'll let Kingman answer that question.

Strohl: There are familial clustering of patients with apneas. If you look at a general population, the estimate is that approximately 40-50% of apneic activity in the population can be accounted for by familial factors—that is, not all of it, but some of it. That's compared to an estimate for asthma would be around 20-25% of all asthma has a familial component; diabetes, about 30-35%. So, there is a familial component. It's probably not unique, and some of the studies that we do of families in Cleveland suggest that there may be 4-5 different ways families express their apneic activity.[1] There is a familial component. That's the level, as to what gene or what process or what intermediate point there is, we're not at a point to understand that.

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McIntyre: I knew a family whose three sons I thought were killing themselves with forks. In retrospect, they possibly could have been victims of familial factors. All were huge men, and there is only one survivor.

Strollo: Recognition can be a factor here. You find patients who have right heart failure out of proportion to their spirometric defect on pulmonary function testing, and people who have right heart failure that persists despite the institution of nocturnal oxygen. Those are patients in whom you should consider the diagnosis OSA. The problem is that there are no great studies that actually tease this out.

SEE PAGE 342

For a listing of sleep organizations in America

Sleep Testing: Center- and Home-Based

Pamela Minkley CPFT RRT RPSGT

Background & Introduction
Polysomnography
  Birth
  Present-Day Description
Limited Polysomnography
  Inconsistency in Validation Results
  As an Alternative to Polysomnography
  Considerations for Use
In Summary

Background & Introduction

The field of sleep medicine and technology has experienced exponential growth since the middle of the 1980s. Recognition of obstructive sleep apnea (OSA) as a pathologic entity and the willingness of third-party payers to reimburse for sleep studies has led to increased development of instrumentation to study the sleeping patient. The National Commission on Sleep Disorders Research, the National Sleep Foundation, and several patient advocate groups have increased awareness of the consequences of undiagnosed sleep disorders, further stimulating growth. The relative ease with which the evaluation and diagnosis of OSA are perceived to be accomplished has enticed many professionals to become active in the testing of sleeping human subjects. Estimates of the total number of sleep centers, sleep laboratories, and other sleep testing facilities in the U.S. range as high as 3,000.

The consequences of undiagnosed sleep disorders, especially OSA, can be so severe that there is an urgency to study as much of the population as possible. Demands for low-cost diagnostic testing have increased quickly at a time that technologic advances are experiencing similarly steep growth. The result has been widespread availability of sleep testing devices without a corresponding growth in medical and allied health profession education in sleep medicine and technology. Research, particularly outcomes-based research, has been unable to evaluate the need or validate the function of these devices at the same rate that they are becoming available. Practice parameters and standards for polysomnography and portable sleep testing have been published but may not be widely applied.[1-4] In my experience, sleep-center-based polysomnography is considered the gold standard for sleep study data acquisition, scoring, and interpretation. Alternative, less costly diagnostic devices may be appropriate for some patients. Cost effectiveness can be achieved only if patients are evaluated clinically and studied on a device appropriate to their particular diagnostic needs and receive adequate education, therapy, and clinical follow-up.

The American Sleep Disorders Association (ASDA) Standards of Practice Committee published practice parameters and standards for portable recordings in the assessment of obstructive sleep apnea in 1994.[3,4] More recently the ASDA polysomnography (PSG) task force has published indications for polysomnography and related procedures.[1,2] The two related documents present an exhaustive search of literature that informs the reader of indications for polysomnography or one of its variations and patient selection for many diagnostic categories. Rapid growth of the technology and the fiscal restraints experienced in sleep medicine and technology today have led to wide use of various alternatives to attended center-based

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polysomnography. Is the technology being applied appropriately, safely, and cost effectively? The purpose of this paper is not to recreate the excellent work of the documents noted. The purpose is to look at recent literature, abstracts, and current practices to detect issues relating to the selection and use of center-based polysomnography versus home, portable, and ambulatory recordings.

One difficulty identified early in preparation of this presentation was the inconsistency of terminology relating to sleep studies outside the sleep center. Figure 1[3] defines some of these terms, and I use these. Portable, home, and ambulatory recordings are differentiated as are attended versus unattended studies. Sleep studies outside the traditional sleep center with less data acquisition capability than polysomnography are all referred to as "limited PSG" unless further differentiation among portable, home, and ambulatory is essential to the discussion.

I combined the results of interactive searching of PubMed, discussions with colleagues, and written comments from participants in educational courses over the past year to identify topics and literature for discussion and investigation.

Polysomnography

Birth

A group of researchers at Stanford University had studied subjects with insomnia and narcolepsy, through the 1960s and early 1970s. When Christian Guilleminault joined the group, respiratory and cardiac sensors not commonly used were included. The resulting sleep study consisting of multiple channels of data during sleep was termed "polysomnography" by Jerome Holland in 1974.[5]

Present-Day Description

Polysomnographic testing today rarely consists of fewer than 12 channels of data, routinely consists of 15-18 channels, and may include 32 or more channels.

Essential physiologic variables[6] and additional measurements or observations that may be useful during PSG are listed in Figure 2.

Definitions

A. **Recording:** Passive collection and storage of physiologic and behavior signals.

B. **Attended:** Personnel are physically present throughout recording session (data observation via modem link is not considered "attended").

C. **Unattended:** Trained personnel are not physically present throughout recording session (data observation via modem link is still considered "unattended").

D. **Monitoring:** Recording of physiologic and behavior events with continuous data observation by trained personnel present either at the recording site or at a remote location.

E. **Portable recording:** Recording that uses moveable equipment that is easily transported for use outside of the sleep laboratory.

F. **Home recording:** Portable recording in the patient's home setting. ("Home monitoring," although often used in a synonymous fashion, technically requires supervision.)

G. **Ambulatory recording:** Recording conducted in a manner that allows a patient to walk around and engage in other usual daily activities during data collection. Although portable equipment is usually used, the patient may be connected to nonportable equipment by direct or radio linkage.

H. **Standard polysomnography:** Polysomnography for the evaluation of sleep apnea, performed under supervision in a laboratory setting, utilizing the measurement of sleep stages, airflow, respiratory effort, oxygen saturation, electrocardiogram, and body position, and the recording of optional parameters such as limb movement, vocalization and carbon-dioxide level.

I. **Screening:** A study conducted on an individual who is asymptomatic for features of obstructive sleep apnea (though possibly at high risk for this disorder). Such studies are most often reserved for epidemiologic and research protocols and are only used clinically when the cost/benefit ratio is low.

J. **Raw data:** Data that are collected and can be reproduced in a format identical to, and visually indistinguishable from, analog data as collected and displayed in a standard polysomnogram. Analog data may be converted to digital form as long as sampling rates allow reproduction to analog signals with sufficient accuracy to allow the same visual interpretation as is possible with the pure analog signal in standard polysomnography.

K. **Processed data:** Sampled, averaged, filtered, staged, or otherwise altered data that cannot be reproduced in a format identical to, and visually indistinguishable from, analog data as collected and displayed in a standard polysomnogram.

L. **Unprocessed data:** Raw data whose only processing is that considered to be routine on standard polysomnography (such as high- and low-frequency filtering appropriate to recorded individual parameters).

M. **Full disclosure:** Presentation of all raw data from a complete study for visual review.

Fig. 1. Terminology relating to sleep studies outside of the sleep center. Adapted from Reference 3.

The ability to observe, record, and analyze multiple physiologic variables individually and in relation to each other on a time line has led to an understanding of circadian variability in brain waves during rapid eye-movement and non-rapid eye movement sleep, breathing and cardiac patterns, and muscle activity during sleep. The studies are dynamic, and patients are closely monitored during the recording period. Technologist-patient interaction and intervention are allowed during the study provided the need for proper assessment outweighs the consequences of waking the patient. Treatment interventions may occur during the study for patient safety or as a cost-savings measure once diagnostic criteria have been met. The studies require attendance by skilled personnel with specialized technical and clinical knowledge. The instrumentation is costly and the typical sleep center consists of two to four bedrooms equipped with polygraphs and audiovisual equipment. Polysomnography is a low volume-high cost procedure. Recent advancements in miniaturization of transducers and data acquisition equipment, digital acquisition and storage of data, development of computer algorithms for com-

Question	Examples of Possible Answers or Considerations
What is the reason for the study?	• Initial diagnosis • Evaluation of current therapy • Screening
What length of recording is required by standards or is necessary to obtain an accurate diagnosis for a patient in this age group with these diagnostic and/or therapy questions?	• Nap • > 6 hours but < 24 hours • > 24 hours • Usual sleep period plus sleep latency or awake time studies
Recording of which physiologic and behavioral variables is essential to adequately represent this patient's sleep?	• CPAP or airway pressure • Central and occipital referential electroencephalography (EEG) • Right and left outer canthus electro-oculography (EOG) • Submental and limb electromyography (EMG) • Lead-II electrocardiography (ECG) • Respiratory effort by 2 or 3 methods • Oral and nasal respiratory airflow, recorded individually or integrated onto one channel • Blood oxygen saturation by pulse oximetry (S_{pO_2})
What additional measurements or observations may be useful?	• Bipolar EEG channels (≥ 8 channels) • Esophageal pressure and/or pH • End-tidal or transcutaneous CO_2 • Positive airway pressure therapy mask pressure • Blood pressure • Body temperature • Actigraphy • Pupilometry • Supplemental EMG channels • Body position • Presence or absence of snoring
Is behavioral observation necessary?	• Can be direct, remote, or recorded
Is there potential need for intervention by trained personnel for patient safety, diagnostic information gathering, or therapeutic intervention?	• Must consider all diagnostic and therapeutic possibilities.
Can the patient attach/detach transducers and start and end the study?	• Must consider the risk of lost or artifactual data in the hands of this patient in this patient's environment.
What instrumentation is available that meets the criteria discovered from the above questions?	• Single channel • Multiple channel (configured for the patient's needs and diagnostic requirements • Video, audio • Ability to be controlled remotely
What environment meets the needs determined for this patient?	• Home unattended, unmonitored • Home, unattended, monitored • Home, attended, monitored • Controlled out-patient environment, unattended, unmonitored • Controlled out-patient environment, unattended, monitored • Sleep Center, controlled environment, attended, monitored by fully trained personnel.

Fig. 2. Questions one must ask and answer in order to choose the assessment method and environment which will best match patients diagnostic and treatment needs. Refer to References 3, 4, and 6 for detailed descriptions and requirements.

puter analysis of data, and improved data transmission techniques have enabled development of options to PSG.

Limited Polysomnography

Inconsistency in Validation Results

Systems and transducers have been evaluated as substitutes for or augmentation to traditional PSG, with inconsistent results for validity, sensitivity, specificity, usefulness as a diagnostic tool, and cost effectiveness. Validation studies of the systems may have considered only function of the equipment or comparison of scored data with the standard PSG, without comment on training of personnel, patient selection, or patient education. All these factors may contribute to the outcome.[7,8] Validation of these systems has often been limited to a specific diagnostic category, usually OSA,[9-11] which may contribute to inconsistency of results.[12] The reader must consider whether the same results would be achieved in other diagnostic categories. The adequacy of training in the use of the equipment and quality assurance measures may not be addressed in the publications. One abstract reported that 70% of the home studies with limited PSG reviewed had data loss.[13] Others have found full PSG in the home reliable.[14] Adequate training of personnel in the use of equipment and scoring of data and the presence of a continuing quality improvement program could account for the difference in the ability to use the systems successfully outside the sleep center. Woodson et al[15] showed that validity could be improved when studies were scored by more experienced technologists. Some limited PSG systems are set up in the home or other environment by a trained technologist. Others are set up in the sleep center, and the patient applies additional transducers and begins the study at bedtime in the home. The distinction between these two methods is rarely addressed in validation data. Comparison data of the two methods are not available.

Patient selection for ability to tolerate transducers and determination of the diagnostic categories appropriate for the device used may also play a part in inconsistent validation of systems. Three different reduced montages were compared in one study[16] with good sensitivity and specificity only when patient selection was appropriate for the montage chosen. Mendelson[17] showed that up to 25% of patients may be misdiagnosed if screened only for sleep-disordered breathing as suspected from the clinical history.

Compliance with positive airway pressure (PAP) therapy has been associated with patient education and follow-up.[18,19] Outcome-based validation of limited PSG systems may be influenced by the amount and quality of patient follow-up versus proper selection of diagnostic equipment—yet, this aspect is rarely addressed in the data.

As an Alternative to Polysomnography

What data are necessary for diagnosis of sleep disorders outside the sleep center setting? Limited PSG is rarely recommended for diagnoses other than sleep-disordered breathing (SDB).[1-4] Actigraphy has been evaluated for identification of sleep/wake state in subjects with insomnia and circadian rhythm disturbances.[20-22] Twenty-four hour ambulatory EEG could be considered a limited PSG in some diagnostic categories because respiratory and EMG data are not recorded, but in this section I deal primarily with methods to diagnose SDB.

Today, sleepiness is widely recognized as a symptom that requires recognition and treatment.[23] The EEG arousals associated with upper airways resistance syndrome (UARS) are becoming recognized as a factor in sleepiness.[24] Limited PSG that cannot detect EEG arousals may fail to reveal the possibility of this diagnosis. Sleep-center-based split-night PSG with PAP therapy initiated midway through the study when diagnostic criteria have been met has decreased the number of PSG evaluations necessary for diagnosis and treatment of SDB in the laboratory from 2 nights to 1 night and, thus, probably represents a significant cost savings. While outcome studies vary, generally the split-night protocol produces compliance with therapy similar to the full-night PSG.[25] The ability to reduce in-laboratory visits by one half has decreased the cost of diagnosis and treatment initiation for SDB, and, I believe, brings into question the cost effectiveness of limited PSG for screening. Patients with a high likelihood of OSA may add cost to their diagnostic work up when screened in the home and then studied again for titration of therapy rather than being studied in the sleep center with a split-night protocol. Self-titrating PAP devices may decrease need for center-based PSG for some patients,[26] but use as a limited PSG diagnostic device has currently (1998) not been completely validated. However, one study has shown one device to be superior to oximetry alone and a promising device for diagnosis of SDB following full clinical evaluation of patients considered for this form of limited PSG.[27] The simplest and seemingly most cost-effective diagnostic tool may be the pulse oximeter. Studies have validated scoring of oximetry strip-chart tracing against PSG for diagnosis of OSA.[28,29] Other studies have investigated alternative methods of analyzing the oximetry waveform.[30-32] Patient selection is key to successful use of this home screening device,[33] and arousals associated with UARS would not be detected. A single channel of airway pressure recordings has been used and validated against PSG for identification of SDB syndromes.[10] The use of a single-channel screening device for follow-up for patients previously diagnosed and treated for SDB syndromes whose symptoms have not been alleviated has not been widely investigated. The need for diagnosis or assessment of current therapy should be a consideration in patient selection for any limited PSG device or system.

Limited PSG for diagnosis of OSA is discussed thoroughly in the ASDA Standards of Practice for Portable Recordings in the Assessment of Obstructive Sleep Apnea.[3,4] Limited PSG devices that include sensors for recording cardiopulmonary variables usually consist of 2-6 channels, including respiratory airflow and S_{pO_2}. They may include respiratory effort, ECG, body position, respiratory sounds, or actigraphy. Sensitivity and specificity increase with the inclusion of additional sensors, clinical questionnaires, and clinical evaluation.[34]

Considerations for Use

Most of the literature that I retrieved addressed the adult population. Clinical evaluation alone in the pediatric population may lack specificity,[35] necessitating full PSG.[36] The validity and cost effectiveness of infant and pediatric evaluations with limited PSG need further investigation. Although unattended limited PSG studies have been investigated,[7,9,11] the impact of technical and clinical intervention available in center-based, attended PSG is not clear. Limited PSG at home is rarely attended due to obvious cost constraints. One study has validated unattended full PSG.[37] Patient evaluations of the experience favored the attended study. They preferred the equipment and availability of a technologist and perceived better quality of sleep in the laboratory.[38] The reasons for the center-based, attended study preference deserve further investigation. Of patients receiving limited PSG testing questioned by telephone interview, 51% reported dissatisfaction with the study; 44% did not review the study with their physician; and 56% were using no treatment although 61% had received a diagnosis of OSA.[39] The availability of PSG equipment for both limited and full PSG with remote monitoring capabilities may offer cost-effective compromise to attended studies. Technologists frequently need to intervene with patients, who are using PAP therapy for the first time or following other therapeutic interventions, to adjust mask fit and eliminate mouth leaks.[40] A fully staffed sleep center may remotely monitor patients in special care facilities or satellite sleep laboratories and direct on-site-trained personnel when intervention is needed.

The rapid introduction of less complicated, more portable alternatives to PSG has led to increased usage by a wider range of personnel outside sleep disorders medicine and technology. Although the initial cost of performing a study may be less, consideration must be given to the overall financial impact if technical quality is poor and clinical evaluation and follow-up are absent or if treatment is not initiated due to false negatives or lack of patient follow-up. The lack of education within the medical community about sleep disorders has been documented by the National Commission on Sleep Disorders Research.[23] The absence of education in sleep and sleep disorders in U.S. medical schools[41] and the low frequency at which sleep histories are obtained in primary care practices have been described.[42] The Walla Walla project[43] describes a significant increase in referrals for sleep-related diagnoses following physician education. The lack of technologist training in sleep medicine and technology is described in another presentation from this conference. The lack of well-trained physicians and technologists to adequately assess, select, perform and interpret studies, and select appropriate treatment options may limit the diagnostic and fiscal success of limited PSG. The Miller abstract[39] raises concerns about whether patients studied in the home receive results of their study or appropriate therapy. The need for education and follow-up of patients on PAP has been established, but information about the amount of education and follow-up available to home study patients is lacking. PAP therapy that is indicated and prescribed but never used results in an untreated patient and an increase in health care costs. Costs may increase also if a co-existing sleep disorder contributing to excessive sleepiness goes unrecognized secondary to poor patient selection for limited PSG, failure to recognize the possibility of co-existing disorder from clinical history or limited PSG data, or lack of patient follow-up. ASDA PSG and portable PSG practice parameters and Canadian PSG standards[1] recognize the need to provide specially trained medical and technical personnel to accurately match the disorder(s) being investigated with the variables being recorded. Figure 2 contains an illustration of issues for consideration in selecting the appropriate diagnostic testing method.

In Summary

Sleep disorders are extremely prevalent in the U.S., and sleepiness resulting from undiagnosed or untreated sleep disorders is responsible for a major cost burden to the American public. The need exists to find cost-effective methods to diagnose these patients. As smaller and less complex diagnostic devices are developed, it is even more likely that physicians and technologists with almost no training in sleep medicine and technology will use these devices. While the initial cost of diagnostic testing may be less than full center-based PSG, the ultimate cost may be much higher if patients:

- are not appropriately matched to the diagnostic testing device and method used;
- do not receive adequate education and treatment follow-up;
- do not receive accurate scoring and interpretation of recorded data or recognition of co-existing disorders.

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Discussion

Mathews: What do the programs represented here do in terms of preparing or, at least, exposing students to sleep diagnostics?

Minkley: Tomorrow we will talk about that a little bit more when we look at some of those training issues. With the rapid growth of sleep disorders medicine, we can't keep up educationally. Education is much slower to move than the initiation of the therapy and the information's getting out to patients. Someone mentioned his first sleep apneic. I remember clearly how in the 1970s, late 70s, we got our first narcoleptic patient, and it was a patient coming in with a *National Inquirer*, showing us how he could be cured by taking a medication called "Gamma." He was sure of it, and it took us a while to catch up with that 'science journal,' his *National Inquirer*, to get a clearer idea of what his perceptions were.

Brooks: You have raised interesting questions, and I hope you don't hate me if I try to pin you down a little bit. On one of your slides, you gave an emphatic "No" with an exclamation point to the question of Does everyone need a polysomnogram? Who does and who does not, and how do we tell?

Minkley: I think the whole issue of identifying patients who are in need of diagnostic evaluation is not clear. We, experts in the field, cannot even define criteria ourselves, so how can a primary care practitioner with very little education in sleep disorders medicine find and select those patients appropriately. Certainly, it would be exclusionary if we were to say every accredited sleep center around or every sleep specialist should require that any diagnostic tool used to assess sleep or sleep-disordered breathing be used only if ordered by a specialist in sleep. That's not realistic. But how do we get the education to keep up with the need? I don't know. You might have some answers. In researching this, I really did come up with absolutely more questions than answers, and I really felt overwhelmed.

Brooks: Well, I'll tell you what. For the sake of discussion, let me say that I think that every person in whom sleep apnea is suspected ...

Minkley: (Now remember, the term was 'sleep-disordered breathing.' I was to look at sleep-disordered breathing.

Brooks: OK. I like that even better.) ...that any patient in whom we strongly suspect sleep-disordered breathing, should have a polysomnogram.

Minkley: How would you define 'strongly suspect?'

Brooks: I would say someone who presented with snoring and something else, and that something else would be signs of daytime sleepiness or witnessed apneas or even someone who's snoring so loudly that it is driving his wife to the couch.

Minkley: I think earlier Dr Chediak had a comment about snoring as an indication for study and saying How do we evaluate that? Do we only look at physiological consequences? I understand that Guilleminault and his group now have looked at cardiac echoes during sleep and some of the changes caused just by simple snoring, and there may be a physiologic impact. But what about the social impact? Again, we aren't used to incorporating the 'soft' signs and issues into the use of diagnostic tools. If a patient has had a spouse or bed partner move from the bedroom because of snoring, but we as sleep specialists listen to the snoring and say "That's nothing! That is nothing! These are just little soft, cute little breathing sounds," then what? If the bed partner is disturbed and moves from the bedroom, how do we give that significance? Is it significant? How do we roll that into the assessment?

Stoller: I'll put on my clinical epidemiology hat for a minute and try to marry two themes and be a little provocative. One is that the diagnostic criteria are uncertain, the population prevalence is uncertain, and it's very difficult to actually come up with the standard diagnostic measures, sensitivity and specificity, positive and negative predictive values, in an unselected population. On the other hand, the other theme I've heard is that sleep apnea is not just a disease that necessarily affects the patient him- or herself, but others in society. When one joins those two issues, it seems to me that the fundamental question is not one of educating the public, but of conducting some population-based study that will ascertain the answers to those questions. What are the common signs and symptoms? What are the predictors of clinical disease and clinical correlates? If, in fact, this line of reasoning is correct, Why is there not a population-based study of this phenomenon? Is it expense? Is it the difficulty of arranging this? Is it disinterest at the level of the NIH [National Institutes of Health]? These are issues that we grapple with in other diseases—α-1-antitrypsin deficiency, for example. There are often specific answers to what the impediments are, so I'm interested in some discussion of whether the proposal is outlandish, and if it isn't outlandish, why there isn't a population-based study that allows these answers to be ascertained?

Minkley: The reasons it hasn't been done is exactly the topic I was assigned—the inability to agree on a diagnostic tool to make that assessment that would be cost-effective to use on large numbers of people. Polysomnography gives us the most information but may not be cost-effective to use, although in the Sleep Heart Health Study[1] they are using full polysomnography. We hope that will bring more information.

Phillips: Dr Stoller's question is to me the critical issue here. I think if we just remain calm, this issue will resolve. Two studies that I know of are trying to address this. One, of course, is the Sleep Heart Health that we've talked about—not really designed originally to investigate sleep and breathing disorders, but containing enough sleep and breathing variables that we'll be able to draw some conclusions. Over 6,000 people have been screened now. So, the work is underway to answer many of the concerns you've raised. The problem with that study is that it is going to be primarily older people, I think. There is also the so-called Wisconsin Cohort, Terry Young's group.[1,2] I don't remember what the denominator is, but they are following 600 very closely and testing extensively. It was designed to answer the question that you posed. So my view right now is that we're in a temporary state of limbo. I think it is important to realize that we are in limbo and to avoid talking about sleep apnea as if we know what it is.

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Minkley: I think you cannot eliminate community education, either because lay people are being 'educated' or at least alerted. In newspapers, sleepiness and its consequences are a frequent presentation. Look at the television shows, the talk shows that deal with sleep apnea. It's kind of dramatic to look at what we call a real 'flaming apneic,' and then cure them. It's showing up on situation comedies. It showed up on *e.r.* The community is being educated in various ways, or at least alerted, and then they are coming to the medical community that may not have had the degree of education they need to answer their questions.

Chediak: I look at the sleep-apneic population in a different way. You have one group of individuals who are impaired in their function during the day or impaired in their physiology while awake, and that can clearly be explained by sleep apnea, not just because it's present but also because when you treat the sleep apnea, the physiologic or functional impairment disappears. Then I look at another group of sleep apnea patients, who may or may not have similar indices of sleep-disordered breathing, as currently gated, if you will, and when you treat that population, you think you're doing a good job, but they remain as functionally or as physiologically impaired as before, suggesting that sleep apnea is not the cause of the diurnal dysfunction. That may be a very innocent way of looking at the problem, but it solves a lot of clinical decision making for me. The ones who do well, I continue to treat, and the ones who don't do well, I rethink the problems and see where I made a mistake in my logic the first time around. Those who get better, we know about those. We're not in limbo about them. We know they have, as Kingman (Strohl) said very early on in the course here today, severe sleep apnea for which you would intubate or you would treat some noninvasive way, and you wouldn't waste much time. Now those are easy to identify and those are well identified. It's the other group we have more trouble with. The other comment I wanted to make was about the expense of all this nonsense that we do to diagnose sleep-disordered breathing. To me, the only way to save a meaningful amount of money is to perform testing outside of the laboratory. Once you're in the laboratory, it makes absolutely no sense to me to do anything less than a polysomnogram. It costs no more money—or very little more money—to do multiple channels of recording when you still need the bed, you still need the machine, you still need the technician, and you still need the ink, than to do maybe a few less channels of recording. This notion is evident in HCFA's [Health Care Finance Administration] approach to reimbursement for a "sleep study," which is what they call 3 or more parameters of breathing with oximetry, etc, but with no recorded sleep, as opposed to polysomnography. The differences in the reimbursement are very small because the way they look at it is, you use the same polysomnogram machine, technician, and laboratory for both tests. The only difference between a sleep study and a polysomnogram is a couple of channels.

Minkley: The question might be "Do sleep professionals, especially when they receive a patient for evaluation, have any obligation to do a 'physical exam' of their sleep, and to what extent and what would that include?" Do you have some obligation to try to rule out anything that might be there? How often is a sleep apneic counseled on sleep hygiene issues? A lot of them come in as sleep hygiene cripples; their sleep patterns have been totally disrupted, and we don't really give them a lot of help to get their sleep back. I think, as was alluded to a little earlier, some of the apneics who get treated indeed come back to their physicians complaining of insomnia because they tried to return to their same sleep-wake patterns, and they no longer need the amount of time in bed that they needed prior to treatment. It may be that their assessment of good sleep was the fact that they used to be able to sit down anywhere and fall asleep immediately. Now that they are treated, when they get into bed, it takes them a few minutes to fall asleep, which is normal, and they interpret that as insomnia. If they're coming to a sleep specialist, that probably would be dealt with adequately; if they are going to a primary care physician, who has had little education in those issues, I'm not sure what the response would be. That's where there's some level of concern—with no answer on how to solve it.

Strohl: Can we get back to polysomnography and the options to polysomnography. First of all, in the Sleep Heart Health Study you don't compare instrumentation between the home and the laboratory because you use the same instrumentation for the patient in the home as you do in the laboratory. So, the issues of technology now are which channel, what monitor, which thermistor?

Minkley: So you're saying it's not an abbreviated ...

Strohl: That's over with now. I think that's over with. So, the next question is, the issues that have been raised by portable monitoring. They both seem to be site-related—that is, is it more appropriate to diagnose and treat patients in centers or in their homes? There are technical issues. There are clinical decision making issues. There are information sources for education in the patient, and there's outcome. The problems and the criticisms of portable monitoring can be turned around and looked at for polysomnography. I predict that in the next 2 years, the value added of having a technician will be questioned. What is the added value of having CPAP titration done in a laboratory? What is the added value of that particular procedure? To me, the challenge is no longer portable versus center-based monitoring, but what clinical and educational resources will be in a system of care. In any system, the more knowledgeable people you have, the better they are at communicating with patients, the more they're able to teach (with different learning styles), the better that outcome will be. But we're talking here about theoretical issues that need to be looked at and critically assessed. I think 10 years from now the sleep lab probably will have to increase its level of expertise and complexity so that it no longer gets paid for doing CPAP titration and filling up half the beds by doing things that maybe can be done more efficiently in other places. So, I think that the debate about the value of the portable monitoring in the last 4-5 years has been helpful to define issues applicable to polysomnography or to a "blood test for apnea" or to any other technique. The fact that portable monitoring is getting slammed really reflects that we have no idea of the cost effectiveness of polysomnography. I have no data from quality assurance programs and outcome from accredited sleep centers, other than some work by Coleman et al in 1983. We know how many people 1 year after a polysomnography believe they benefited from that procedure—that it was worthwhile. So, I think that these are generic issues. To me sleep apnea occurs in the home, it occurs in the bedroom in which the spouse is absent, and you treat it in that area. Titrating a person in the laboratory and having him go home and having it not work is just as bad as titrating him at home—and maybe even worse. There are some real issues that need to be addressed by the field. These are recognition, appropriate treatment, including very simple things to treat, that primary care practitioners *can* do, that HMOs *can* do, and that health care systems can do. We need to help the primary care practitioner get over our current blanket recognition of the need for an expert or expert laboratory for all patients. I think we have to be more proactive in setting up those systems.

Minkley: I absolutely agree.

Atwood: Even more practically, how does the issue of reimbursement fit into all this? If we can't get paid for doing a home study or the reimbursement is too low to support everything else that needs to go on in our programs, how will we make them viable parts of our sleep labs?

Minkley: This is the issue, again, of education, and the component of education that frequently is left out is being proactive in educating HMOs, PPOs, and third-party carriers. Certainly that education curve lags behind the availability and use of technology. So we're constantly weighing the needs, the technology available, the reimbursement available, and indeed, the criteria even to get the treatment modalities paid for. While I think there was some degree of consensus in this room that there are patients at whom we can just look and identify as sleep apneics and start on nasal CPAP, if we don't have a number for the RDI or a number of apneas to supply to an insurance company, they don't want to pay for their therapy. So, it's a difficult situation.

Brooks: Let me disagree with many of the things that the last couple of speakers have said. Number one, I cannot identify an individual in whom I do not want a polysomnogram. I can't tell that this guy is certainly going to need treatment, and I can't tell what treatment this person is going to need without the help of an in-laboratory polysomnogram. I will show some data later that suggests other people can't either. On the issue of in-home versus in-laboratory, the technology is great. The sensors are small, they're portable, they can be put on. They fall off. You described an incident where the wife had the oximeter on her. That study was wasted. We are dealing with a potentially life-threatening problem that does not have simple solutions. The treatments that we have to offer range from cutting your throat to wearing a silly mask over your nose for maybe the rest of your life. If those are the options that my physician is offering me, I want him or her to be damned sure that that's what I have and that that's what I need. I don't want somebody who is a pulmonologist who attended a course prior to the ATS meeting and who has learned a little bit about CPAP to say "You snore. Here's a home CPAP device." There are lots of other things that I may have. There are other treatments that may be warranted. Similarly, if I happen to take the same symptoms to an otolaryngologist, I don't want surgery. I want somebody who's going to consider all of the treatment options that we have, and the best person to do that is somebody with expertise in sleep medicine and access to an accurate diagnostic assessment of what's wrong with me. I think that a lot of what we are doing has been pushed by the perceived need to cut costs. Well, I think that good medicine is cost-effective. You find a disease, you treat it, and the person is not going to crash his car or do poorly in school or at work or whatever. People sometimes refer to limited studies as screening studies. I think that a screening study is appropriate if you're going out to the GE plant to study 500 people, and you want to learn something about breathing or sleep. I think that when a patient presents to you as an individual physician, you are not engaged in worrying about the health of the community at large. You're worried about the health of this individual in front of you, and the only way to really determine that is with a polysomnogram.

Erman: As someone coming from the hothouse of managed care, I'll offer a few comments and speak more about this tomorrow. I would also disagree with some of what we have heard, but perhaps from a slightly different perspective. There is an illusion of cost saving in the home and an illusion that this is a more appropriate environment, which is not necessarily the case. Limited studies are of limited benefit, and, in fact, it may very well be that the most effective use of technology and personnel is, after appropriate screening, use of a split-night study attended in the sleep lab, that allows confirmation of diagnosis, initiation of treatment, and quality control in terms of use of nasal CPAP. I would also say that contrary to the experience in other parts of the world, it may very well be that for certain managed care payers, initiation of nasal CPAP without a study may be perceived to be a *very* effective use of resources. In fact, there is one protocol that is being used—or abused, perhaps—in certain managed care settings. Based on limited studies, there's a presumptive diagnosis of sleep apnea. If treatment is tolerated, and, with use of techniques such as an oximetry, there is an evident remediation of pathologies, it's considered a good outcome, and the cost of a study is avoided. It has not been well validated, and is certainly not as gratifying from a scientific perspective, but if one simply looks at a cost-benefit basis, it may be a supportable approach.

Minkley: With some of these other diagnostic tools, the question is Is there a knowledgeable person following that patient to assure the results are applied appropriately? If that particular tool produces a negative result, what happens to that patient? Because, again, it is not cost-effective if it is used and then a negative study is just accepted as negative. The patient is still symptomatic. That has not been dealt with and may ultimately cost the system money.

Phillips: It is not uncommon for me to see a patient like the one 2 weeks ago, who was obese, indigent, unemployed, a cigarette smoker, and between jobs for about 10 years. He had no insurance but he did have a wife, who knew he had sleep apnea—and so did every one in my clinic from the janitor to me—350 pounds, sleeping in the waiting rooms, snoring sitting up kind of thing. It was clear that he had sleep apnea, the kind that Kingman (Strohl) and I would treat tonight, not tomorrow. He had no money for a sleep study and no money to pay for CPAP. Now, I don't know how others in this room would handle it, but we rotate home care companies to the clinic, and the one that was there that day offered to lend or give him a used CPAP machine, and we set him up on CPAP. Then the issue became how much CPAP. So, I used that very classic and widely used clinical algorithm, called "what the heck, half the neck." It always works out to about 10 cm H_2O, give or take 2, and at the risk of starting everybody else on another tirade, I would say that empiric CPAP is extremely cost-effective, and based on studies about night-to-night variability and need and CPAP and what happens to people who have been treated with CPAP who come back and are retitrated 2 weeks later and so forth, I personally believe that the value of a CPAP titration is over-rated. Now I'm going to climb under the table right now, but I do think that's true. Now, this patient has already called me once to thank me and tell me that he's feeling better, and I think it will work. So, I support what Milt (Erman) said. There are patients whom I would treat without a study, but maybe for a different reason, and they don't necessarily have to be in the ICU.

Brooks: If that same patient had come in and had had a job and had insurance, would you have done the same thing?

Phillips: Of course not. Because his insurance would not … it was almost a luxurious situation, right? To get the insurance to pay for CPAP, where I come from, a patient has to have 30 apneas in 6 hours or 30 apneas before you do your CPAP titration, if you're going to do a split-night study. I had the remarkable freedom of knowing and deciding on my own "Yes, this is sleep apnea. Yes, it needs treatment. No, no insurance executive is going to tell me whether or not I can treat it. I can just do it!" It was great.

Epstein: Let's suppose that there are no insurance companies and money doesn't matter. Would you have preferred the patient to have a diagnostic study in the lab? Maybe he had obesity hypoventilation syndrome on top of sleep apnea. Deciding whether to test should not be based on ability to pay. However, determining how best to arrive at that decision is greatly affected by cost in our current environment.

I think it's very interesting how we have changed the discussion. I think this is an important point. Originally we were talking about what test is best for diagnosing sleep apnea. Now we are talking about making clinical decisions based on economic issues. I think it's becoming fast irrelevant whether you test in-laboratory or whether you do it in the home. Advances in technology are driving this change, and the quality of the equipment has improved to the point that you can get just about equal results from either in-laboratory or at-home systems. The considerations that are going to decide which we use now are economic: which test is going to be most cost-effective, which is going to provide the most beneficial long-term outcome, are people going to be more likely to use CPAP if they are titrated in the lab versus the home, and how are we going to pay the increased cost from better recognition of this burgeoning problem? Those are the issues here. The question no longer is whether we use an in-home or in-laboratory study, but how we answer the other questions. So, unfortunately, I think economics has a big role to play in the clinical decision because technology has settled the other question.

Minkley: Do you feel sleep studies should be attended by technicians to improve quality or unattended to save cost?

Epstein: I think technologically you can envision ways of doing both. For instance, you could have an unattended study in which you have somebody monitoring the study in the sleep laboratory by modem. One technician could watch a bank of 20 sleep studies, improving cost-effectiveness, and make sure that leads don't fall off, improving quality. The technology has advanced dramatically over the last 5 years. When we had this discussion 5 years ago, it was oximetry versus in-lab polysomnography. In another 5 years, the discussion will be Is it going to be more cost-effective to use an auto-CPAP machine equipped with a diagnostic chip versus an in-laboratory split-night study versus something that hasn't been developed yet? The questions that I think we need to answer next are, first, which method is most cost-effective and beneficial, and second, which is going to improve compliance with the therapy?

Minkley: We need the data, but, yet, the technology continues to evolve at a rapid pace. A number of well-trained, knowledgeable physicians, PhDs, and technologists are available today to apply some of these correctly, yet the growth seems to be outstripping the ability to train clinicians, technologists, and third-party payers.

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SLEEP-DISORDERED
BREATHING: PART II

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Positive Pressure Therapy: Theory and Application

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Introduction
Pathophysiology of Obstructive Sleep Apnea
Positive Airway Pressure (PAP) Applications
  Continuous Positive Airway Pressure (CPAP)
    Mechanism of Action
    Titration
    Prediction Equations
    Side Effects & Complications
    Compliance with Therapy
  Bi-Level PAP
    Mechanism of Action
    Titration
  CPAP versus Bi-Level PAP
  Technical Considerations
    Interfaces
    Humidification
  Auto-Titrating CPAP Units
    Background & Rationale
    Clinical Trials
    Future Developments
Future Role of the Sleep-Laboratory Technologist
In Conclusion

Introduction

Treatment of obstructive sleep apnea (OSA) has evolved from surgical therapy, such as permanent tracheostomy, which bypasses the site of obstruction in the upper airway, to positive pressure therapies that alleviate upper airway obstruction through air-pressure 'splinting' of collapsible upper airway segments. Since its description in 1981 by Sullivan and colleagues,[1] continuous positive airway pressure (CPAP) therapy has become the treatment of choice for OSA.[2] Although newer surgical procedures are available to treat OSA and experimental approaches to treatment, such as electrical stimulation of pharyngeal dilator muscles are under development,[3] positive airway pressure therapy is likely to remain the mainstay of therapy well into the future. In this paper, I discuss conventional nasal CPAP therapy and other forms of positive pressure therapy, including bi-level positive airway pressure (bi-level PAP), and self-adjusting CPAP devices. In addition, the evolving role of the sleep laboratory technologist in patient evaluation and management is discussed.

Pathophysiology of Obstructive Sleep Apnea

The hallmark pathophysiologic event of OSA is upper airway closure during sleep. Although this concept is simple at first glance, the underlying pathophysiology is complex and

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remains incompletely understood. The most comprehensive understanding of OSA pathophysiology has been developed by Remmers and colleagues.[4] Their "balance of pressures" theory explains how opposing forces of pharyngeal narrowing and dilating create a dynamic balance that defines moment-to-moment pharyngeal caliber. Forces favoring dilatation primarily arise from the pharyngeal dilator muscles,[4,5] the most important of which is the genioglossus. Dilator forces are opposed by "tissue pressure" forces, which favor collapse of the airway.[4] These include the effect of gravity on the upper airway structures,[6] pharyngeal fat pads,[7] body weight,[8,9] and overall neck mass.[10,11]

Pharyngeal compliance, the overall 'stiffness' of the airway, is an important determinant of the potential for upper airway collapse.[12] Studies in awake humans with OSA have demonstrated increased compliance compared to normal subjects.[13] Morrison and colleagues[14] studied a similar group of sleeping patients with OSA and found increased compliance in the nasopharynx compared to the oropharynx or hypopharynx.[14] Important effects of age, gender, and obesity on upper airway physiology have also been described.[15]

The role of upper airway anatomy in OSA pathophysiology has received considerable attention but remains incompletely understood.[15] Clinical studies have consistently found overall neck mass as determined by circumference to be an important risk factor in sleep apnea.[16,17] Pharyngeal fat deposition, particularly in the lateral pharyngeal wall, has also emerged as an important risk factor.[18] Finally, the role of craniofacial anatomic disproportion as a risk factor for OSA has received attention.[19] Whether these craniofacial abnormalities cause OSA or are a result of OSA is not certain. Longitudinal studies of the role of craniofacial abnormalities in the resulting development of OSA are needed to advance this topic of research.

Positive Airway Pressure Applications

Positive pressure therapy is a generic term that applies to several different treatment modalities. The oldest and most widely prescribed positive pressure therapy is CPAP. This treatment may be applied by way of a nasal mask, a nasal prong system, or an oronasal mask (ie, full face mask).[20,21] The defining feature of CPAP therapy is that the airway pressure delivered to the upper airway remains constant through inspiration and expiration.

Bi-level positive airway pressure, first described in 1990,[22] is an alternative to CPAP for treatment of OSA. Bi-level PAP devices differ from CPAP units in that inspiratory and expiratory pressures can be independently adjusted. This takes advantage of the observation that the level of air pressure required to maintain pharyngeal patency differs throughout the respiratory cycle. In general, once airway patency is established, a lower expiratory pressure will maintain upper airway patency. For some patients, bi-level PAP may be more comfortable than CPAP because the patient exhales against a lower mask pressure. Bi-level devices tend to be more expensive than CPAP units.

The most recent option in positive airway pressure therapy is 'smart', or auto-titrating, CPAP therapy. The underlying principle of these units is that the CPAP pressure delivered by the unit varies in response to the amount of pressure required to maintain upper airway patency. These units attempt to limit the amount of CPAP pressure delivered to the upper airway to the minimum level necessary to maintain airway patency at any point during the patient's sleep. The different positive airway pressure therapy options, their year of introduction, and relative advantages are shown in Table 1.

Table 1. Positive Pressure Therapy Options

Mode	Year Introduced	Advantages
CPAP*	1981	Most familiar option Easiest to titrate Lowest cost
Bi-level PAP	1990	Better initial acceptance? Easier to exhale against
Auto-titrating CPAP	1995-1996	Less technician time Potential for home study Lower mean pressure Greater comfort?

* CPAP = continuous positive airway pressure; PAP = positive airway pressure.

Continuous Positive Airway Pressure

Nasal CPAP is the most widely prescribed specific therapy for OSA, used by approximately 85% of patients in whom positive pressure therapy is prescribed.* It is the therapy of choice in the treatment of OSA. CPAP has virtually eliminated the need for permanent tracheostomy for management of OSA.[23] Surgical therapy is now an elective procedure for OSA rather than the established therapy, as was the case prior to the development of CPAP.

Mechanism of Action. The mechanism of action of CPAP is to splint the upper airway via positive air pressure applied through a nasal or oronasal interface.[1] Evidence for CPAP's mechanism of action comes through direct observation of the upper airway during the application of CPAP,[14,24] and from catheter measurements of pharyngeal pressure, which have demonstrated that pharyngeal pressure is reversed from negative to positive during CPAP administration.[1] This positive air pressure splint counteracts the negative intraluminal pressure forces that promote airway closure.[4]

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*Mark Estes, Respironics Inc, 1997: personal communication.

A reflex dilation of the upper airway based on CPAP-induced increases in lung volumes has been proposed as an additional mechanism by which CPAP causes pharyngeal dilatation.[13] This mechanism has been disputed by others.[25]

The effect of nasal CPAP on upper airway muscle tone has also been studied. Several groups have suggested that CPAP inhibits pharyngeal dilator muscle tone rather than augmenting it.[26,27] If pharyngeal dilator muscle tone were diminished by CPAP, one would expect pharyngeal airway narrowing rather than the dilatation that is clearly the net effect of CPAP therapy. The physical airway splinting effect of CPAP appears to be the predominant effect of this mode of therapy.

Titration. It is conventional for nasal CPAP to be prescribed after a sleep laboratory titration has determined the level of air pressure that provides airway patency at all times during the patient's sleep. In this regard, CPAP is titrated to treat the 'worst case' of upper airway closure seen during the patient's CPAP-titration sleep study.

In theory, CPAP therapy can be initiated at any pressure level. CPAP therapy could be initiated at the maximum level and decreased over the course of the study until apneas and hypopneas occurred. However, for most patients, therapy is initiated at a low pressure level and then titrated upward until apneas and hypopneas are eliminated. The usual initial pressure level is 5 cm of water pressure. Lower pressures may be used, but, in our experience, some patients feel 'suffocated' at pressures lower than 5 cm H_2O due to the lower airflow at the lower pressure. Initiation of CPAP titration at higher pressures may be appropriate if the patient has prior experience with CPAP or if a high CPAP level is anticipated. If positive pressure is initiated at a higher level and few apneas or hypopneas are detected, the pressure should be lowered until evidence of upper airway narrowing occurs. A final pressure prescription is determined to be the level at which sleep-disordered breathing is just eliminated. A successful CPAP titration establishes the minimum pressure needed to maintain upper airway patency.

During a CPAP titration, pressure levels can be changed in large or small increments, depending on the severity of the sleep apnea and the appearance of pressure-related side effects. Many laboratories start with a CPAP level of 5 cm H_2O and make subsequent changes in 1-2.5-cm increments until a final pressure level is reached. At the University of Pittsburgh Pulmonary Sleep Laboratory, we begin the CPAP titration at 5 cm H_2O and initially increase pressure in 2.5-cm increments in response to apneas and hypopneas. A change in pressure is made after a cluster of 2-4 consecutive apneas or hypopneas is observed. This approach results in a rapid increase in pressure during the early part of CPAP titration. CPAP titration continues until OSA is significantly reduced, the maximum unit pressure is reached (approximately 20 cm), or the patient is unable to tolerate the pressure. When apneas and hypopneas are reduced, but still occasionally present, mask pressure is increased in 1-cm increments until they are essentially eliminated. This achieves the goal of obtaining optimal relief from sleep-disordered breathing at the lowest level of pressure.

Inspiratory flow limitation and associated arousals may be considered forms of sleep-disordered breathing and are also targets for pressure increases.[28] Although apneas and hypopneas may be eliminated, elevated upper airway resistance may still be present. The increased airway resistance may result in arousals from sleep.[29] Upper airway resistance can be monitored during a polysomnogram by measurements of pleural pressure using an esophageal balloon. As an alternative to an esophageal balloon, a nasal pressure pneumotachygraph system may be used to detect slight changes in the inspiratory airflow signal. This airflow monitoring technique has been proposed as a noninvasive surrogate for upper airway resistance.[28]

Arousals from sleep associated with changes in airflow characteristics of the upper pharynx may be treated with nasal CPAP. However, no standard approach to titrating positive pressure therapy to increased upper airway resistance exists. At the University of Pittsburgh Pulmonary Sleep Laboratory, we increase pressure 1-2 cm H_2O above the level at which apneas and hypopneas are eliminated if we observe inspiratory airflow limitation and arousals. If the arousals are not eliminated, pressure is reduced to the previous level.

Prediction Equations. Another approach to CPAP-level selection is to apply a mathematical formula based on a prediction model of the level of pressure required to treat OSA. A prediction model, based on body weight, neck circumference, and apnea index has been described.[11] One advantage to such an approach is that the final pressure may be more rapidly achieved. Consequently, a longer time during the CPAP titration is spent at the therapeutic pressure level, which increases confidence in the final CPAP prescription. If this approach is used, the prediction formula should be validated in each sleep laboratory's population because characteristics of patients vary among sleep laboratories.[30] Although attractive in concept, this approach has not been extensively tested in clinical trials.

Side Effects & Complications. Side effects are common with CPAP therapy, but medically significant complications are rare.[31] As shown in Table 2, most side effects are related to the mask or other patient interface or the pressure generated by the air blower. Occasionally, CPAP noise is a source of therapy intolerance, but this has largely been eliminated with the much quieter contemporary CPAP units.

CPAP mask-related side effects are usually the result of poor mask fit. Poor mask fit frequently results in air leaks

Table 2. Intolerance of CPAP* and Potential Solutions

	Problem	Possible Solution
Mask-related	Poor fit	Refit mask; tighten or loosen headgear straps
	Mouth leak	Chin strap; humidifier; oronasal mask
	Claustrophobia	Desensitization;try nasal prong interface
Pressure-related	Difficulty exhaling	Try ramp settings; consider bi-level PAP
	Nasal congestion/ rhinorrhea	Nasal decongestant
	Nasal dryness	Humidifier
Other	Air temperature†	Increase room temperature; heated humidifier; put CPAP tubing under blanket.
	Humidity	Water in mask: remove humidifier
	CPAP blower too loud	Move farther away from bed

*CPAP = continuous positive airway pressure; PAP = positive airway pressure.
†Air temperature usually too cool.
(Adapted from Reference 23 and Reference 28)

around the mask into the eye, forehead, or mouth, causing awakenings from sleep. The importance of mask fit and comfort should be emphasized because poor fit may hinder acceptance of the therapy. Patients often develop their own solutions to these problems, such as adding extra padding to one section of the mask or to the contact area with the face (eg, a Band-Aid™ on the bridge of the nose). A knowledgeable sleep-laboratory technologist can assist patients experiencing mask discomfort, correct poor mask fit, and greatly enhance patient comfort. If one mask is too uncomfortable for long-term use, a new mask should be fitted. Some patients prefer to have several masks that can be used in rotation to maximize comfort. This adds extra cost to the therapy but may be necessary to ensure effective CPAP use. Mask claustrophobia is occasionally encountered. This may be successfully treated with progressive desensitization to the presence of the mask on the patient's face.[32] Table 2 shows common CPAP intolerances and possible solutions.

CPAP-related side effects of the mask are a common cause of CPAP intolerance. Although it seems intuitive that higher pressures would be associated with a greater frequency of side effects, studies examining this issue do not support this.[31,33] Many contemporary CPAP units have a 'ramping' feature that allows the patient to initiate CPAP therapy at a pressure lower than the final pressure and then have the pressure gradually increased to the final pressure over a period of 15-30 minutes. This allows the patient to fall asleep while the effective pressure is gradually reached. This feature may be particularly helpful for patients who have difficulty exhaling against the pressure provided by the device.

Air temperature inside the mask—either too cold or too warm—is occasionally the source of patient discomfort. Temperature of the air can be manipulated with a heated humidifier or by changing the air temperature in the patient's bedroom.

Compliance with Therapy. Successful treatment of OSA with CPAP (or other positive pressure modalities) depends on patient compliance. Studies in which patient compliance was monitored by patient report have demonstrated a 65-90% rate of compliance.[34-36] More recent studies in which compliance with therapy was objectively and covertly measured by built-in counters measuring actual 'on' time have found significantly less regular use.[37,38] Kribbs and colleagues[38] found that 46% of patients were compliant with CPAP therapy, using a generous definition of compliance (> 4 hours of CPAP/night on 70% of nights).

Due to conflicting results from research reports, the factors contributing most to compliance with CPAP therapy remain unclear. One study found significant associations between good compliance and the patient's perception of therapy benefit and disease severity.[39] Other studies found no clear associations between regular CPAP use and either objective disease severity or subjective improvement rated by the patient.[37,40] Given the importance of CPAP compliance for optimal CPAP therapy, more work in identifying determinants of compliance is needed.

Bi-Level Positive Airway Pressure Therapy

Bi-level positive airway pressure is an alternative to CPAP for treatment of OSA. First described by Sanders and Kern,[22] bi-level PAP devices have gained widespread acceptance among sleep-laboratory clinicians and patients. Bi-level PAP differs from CPAP in that the airway inspiratory and expiratory pressures are independently adjustable. Inspiratory assistance is provided by inspiratory positive pressure, and expiratory positive airway pressure provides an airway splint, similar to CPAP. As with CPAP units, bi-level PAP units are capable of generating a maximum pressure of 20 cm H_2O.

Bi-level PAP units also function as pressure support ventilators, a role primarily reserved for patients in intensive care units. The use of bi-level PAP for acute respiratory insufficiency was recently reviewed[41] and will not be considered further. Bi-level PAP units, unlike volume-cycled ventilators set in the pressure support mode, are moderately leak tolerant. This allows their use for OSA patients who typically have some degree of pressure leak either from incomplete mask seal or through the mouth. Approximately 10% of OSA patients treated with positive airway pressure therapy use a bi-level PAP device.*

Mechanism of Action. Bi-level devices operate on the principle that a higher level of pressure is needed to open the airway than is needed to maintain its patency. Sanders and colleagues[42] have shown that upper airway pressure becomes slightly negative during expiration, promoting airway narrowing, instability, and possible closure with the next inspiratory effort. They postulated that airway pressure splinting requirements would be different during inspiration and expiration, with a lower pressure being required during expiration. This led to the development of a device that allowed for independently adjustable inspiratory and expiratory pressures. Bi-level devices have been shown to provide effective treatment for OSA.[22,23] In comparison to CPAP, they maintain airway patency at a lower expiratory pressure. Patients may find bi-level PAP devices to be more comfortable than CPAP units.[2]

Bi-level PAP units initiate inspiratory pressure in response to a low level patient-generated inspiratory effort (approximately 40 mL/s).[22,23] When inspiratory airflow drops below a set level, the delivered air pressure decreases to the set expiratory pressure level while the patient exhales. Inspiratory pressure applied to the upper airway offsets the negative intrapharyngeal pressure generated during inspiration. During expiration, expiratory pressure 'splints' the upper airway, preventing closure. Expiratory pressure is titrated to treat upper airway closure, the physiologic correlate of obstructive hypopneas and snoring (primarily heard during inspiration). Inspiratory pressure may also be titrated to decrease or eliminate inspiratory flow limitation and associated arousals.

Titration. The goal of bi-level PAP titration is the same as for CPAP titration: to eliminate sleep-disordered breathing. As with CPAP titration, a standard and widely accepted titration protocol to guide the sleep laboratory technologist is not available. Therefore, sleep laboratories performing bi-level PAP titration have only general guidelines for administering this therapy. Although manufacturers of these devices generally provide educational materials and a generic approach to titrating pressure to meet certain therapeutic goals, these protocols may vary across manufacturers.

The principles governing bi-level pressure titration are similar to those for CPAP. PAP is increased primarily in response to apneas and hypopneas until either they are eliminated or the patient can no longer tolerate the therapy. Because more than one pressure level is manipulated during bi-level PAP therapy for OSA, this titration may be more complicated than those for CPAP therapy.

A general approach to titration can be described. As with CPAP titration, bi-level pressure titration can be started at any pressure. We typically begin at an inspiratory pressure of 5 cm H_2O and expiratory pressure of 2.5 cm. Expiratory pressure is increased in 2.5-cm increments in response to obstructive apneas. Inspiratory pressure is increased in 2.5-cm increments for obstructive hypopneas. In the initial portion of bi-level titration, inspiratory and expiratory pressure may be equal to each other, ie, the positive pressure is continuous at one level. Once apneas are eliminated, the inspiratory pressure is increased further for obstructive hypopneas, inspiratory flow-limitation-associated arousals, snoring, or periods of hypoventilation. Once the technologist conducting the study believes elimination of sleep-disordered breathing is nearly complete, pressure is adjusted in 1-cm increments to 'fine tune' the final bi-level PAP prescription. As with CPAP, the goal is elimination of sleep-disordered breathing at the lowest level of pressure possible.

Bi-level devices may be set in one of several modes, depending on the patient's ventilatory needs and the disorder being treated. For many patients with OSA, a spontaneous mode is appropriate. In this mode, the bi-level PAP unit provides inspiratory assistance only in response to spontaneous inspiratory effort by the patient. A timed mode may also be available but is not necessary in spontaneously breathing patients. Bi-level units also have a mode that combines spontaneous and timed features. Patients with mixed forms of sleep-disordered breathing, such as OSA and central sleep apnea, or marked degrees of nocturnal hypoventilation, such as is seen in patients with neuromuscular and chest-wall disorders, may benefit from the combined mode. In this situation, the spontaneously breathing patient triggers the unit by inspiratory effort as long as such efforts continue. If a central sleep apnea or other ventilatory pause occurs, the timed component provides backup ventilatory assistance. The length of ventilatory pause allowed before the backup rate is triggered depends on the rate chosen, which is set by the sleep-laboratory technologist.

The technologist should strive to reach the final pressures, or close to them, as quickly as possible. I believe that the clinician responsible for the study may be less likely to be confident of the final positive pressure prescription arrived at in the last 30 minutes (or less) of sleep than of that prescription arrived at for the last 200 minutes of sleep. Obviously, early attainment is not possible in all cases but should be thought of a general goal. Sleep-laboratory technologists, managers, and physicians managing CPAP or bi-level PAP therapy should examine this aspect of the sleep study closely to assure therapeutic adequacy.

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*Mark Estes, Respironics Inc, 1997: personal communication.

CPAP vs Bi-Level PAP

Few studies comparing CPAP and bi-level PAP units in the treatment of OSA have been performed. The largest of these focused on the important issue of therapy acceptance and long-term compliance and found better initial acceptance of therapy of bi-level therapy compared to CPAP.[43] Long-term compliance with therapy, however, measured by average hours of use per night, was equal between the two groups at 1 year from the initiation of therapy. In my experience, bi-level PAP units are more expensive than CPAP units and are therefore usually reserved for patients unable to tolerate CPAP. Bi-level pressure therapy, due to its differing inspiratory and expiratory pressures, may be preferable in patients with nocturnal hypoventilation and hypercapnia, although no comparative trials specifically examining this patient group have been performed.

Technical Considerations

Interfaces. Regardless of whether a CPAP or bi-level PAP unit is chosen, the choice of the patient interface is critical for long-term success. Surprisingly few objective data on the importance of CPAP mask choice and comfort are available. This aspect of care is important to patients. An uncomfortable mask is likely to doom acceptance of therapy and long-term use to failure. Proper mask fit has been aided by newer interfaces that appear to be more comfortable than earlier generations of masks. Response to inquiries about mask fit, comfort, and side effects is important information to obtain at each patient visit after therapy has been initiated.

Humidification. Humidification of the air delivered by any type of positive pressure unit should be considered but may not be necessary in all cases. Patients seem to have a wide range of tolerance for the dryness of the air delivered. Seasonal variations in air humidity may affect patient tolerance. For instance, the more humid air in the warmer months may be better tolerated than the dry air of cooler months. Humidifiers are frequently added to CPAP and bi-level pressure units to add moisture to the inspired air.

The humidity provided varies with different types of humidifiers. Heated humidifiers, which warm the inspired air slightly, achieve the highest levels of humidity; however, unheated humidifiers probably are more commonly provided because of their lower cost. The importance of inspired air humidity in CPAP therapy has been discussed by Richards and colleagues,[44] who found that nasal resistance decreases significantly when the CPAP air is humidified. Although patient outcomes trials specifically comparing humidifier use to no humidifier use have not been performed, a large French study[28] found no difference in the incidence of CPAP side effects with respect to humidifier use. Because humidifiers add cost to the equipment purchased, its relative importance in each patient's treatment plan should be considered.

Auto-Titrating CPAP Units

Background & Rationale. The most recent addition to the growing array of positive pressure therapy options are the 'smart,' or self-adjusting CPAP units (or auto-CPAP). These units have been developed by manufacturers and are now widely available. An underlying rationale for the development of these units is the understanding that because upper airway tone and potential for narrowing and closure vary across sleep in all patients, it stands to reason that the amount of positive pressure needed to treat upper airway collapse should vary across the sleep period as well. Conventional CPAP units provide a fixed amount of pressure through the night, but the pressure level needed for maintenance of airway patency may vary as well. It is possible that if the mean CPAP level can be decreased, patient tolerance of and compliance with therapy may increase.

Another explanation for the interest in auto-CPAP systems is the effort to decrease the costs involved in the evaluation and treatment of patients with OSA. Due to the personnel and sophisticated monitoring equipment, sleep-laboratory-based patient evaluations and treatment are time and labor intensive and, therefore, associated with high costs. If some part of the evaluation and diagnostic testing, such as the CPAP titration, could be done outside of the sleep laboratory, such as in the patient's home, OSA treatment costs might decrease. In this regard, the use of self-titrating CPAP units for a CPAP treatment trial at the patient's home holds promise.[45] If this approach can be demonstrated to be safe, clinically effective, and cost-effective, this approach is likely be attractive in our increasingly cost-conscious medical marketplace.

The concept behind each of the different commercially available auto-titrating CPAP units is the same: positive airway pressure is adjusted by a proprietary computer algorithm that detects and responds to some aspect of upper airway physiology. Changes in the 'target' signal result in appropriate changes in CPAP. Such target signals include changes in pharyngeal airflow contour and detection of upper airway vibrations indicative of pharyngeal narrowing.

Auto-CPAP devices adjust CPAP according to an internal microprocessor algorithm. Each manufacturer's device has a pre-set pressure increment and pressure rate of rise that determine how rapidly the auto-CPAP unit responds to apneas and hypopneas and by how much pressure. Each auto-CPAP system monitors changes in upper airway physiology and translates these changes into a likelihood of developing apneas or hypopneas in the next several respiratory cycles. CPAP is adjusted accordingly to treat sleep-disordered breathing as it is initiated.

Auto-titrating CPAP units potentially offer several advantages over traditional units. They allow a greater degree of flexibility in location and timing of the study. Auto-CPAP therapy may be applied now in a variety of settings, ranging from the conventional sleep laboratory, where the study is attended and conducted by a sleep technologist, to an unattended study at the patient's home. They also provide adequate upper airway stability in times of increased instability, such as during use of alcohol or sedative medications.

Clinical Trials. Auto-titrating CPAP units have been studied in a small number of clinical trials,[46-49] with somewhat conflicting results. When auto-titrating CPAP therapy has been compared to a standard sleep-laboratory-based CPAP titration, generally equivalent results have been shown. One study,[48] however, demonstrated inadequate treatment of OSA when the auto-CPAP result was compared to a manual CPAP titration. When mask pressure between standard CPAP and auto-CPAP were compared, another study found that auto-CPAP resulted in a 57 ± 5% lower mask pressure than that found during the standard CPAP titration.[50] It is possible that this amount of pressure reduction over the course of a night could result in greater mask comfort and better compliance with therapy. Although many factors influence patient compliance, comfort with the CPAP unit and interface is likely to be among them.

Future Developments. The optimal use and future role of auto-titrating CPAP therapy is evolving. The technology has advanced to the point now that the conventional sleep laboratory may not be necessary for the evaluation of all patients with sleep apnea. In the future, diagnostic studies may be done with increasing frequency outside the traditional sleep laboratory. These studies will not routinely be attended by a sleep-laboratory technologist. Technology already allows such studies to be performed using an abbreviated form of monitoring (such as a 4-channel monitor) or a miniaturized polysomnograph recording. If OSA is diagnosed, a CPAP titration study may be performed in the patient's home, using an auto-titrating CPAP unit. Clinical trials demonstrating safety and accuracy of such an approach should be performed before such programs are put into place.

One current use for auto-CPAP is in the sleep laboratory as a way to increase the number of patients treated with CPAP through the sleep laboratory. With this approach, the auto-titrating CPAP unit is a labor-saving device; more patients may be treated with CPAP through the laboratory without increasing the number of technicians. Patients at low risk of positive pressure complications and without significant cardiorespiratory disease may be studied in the sleep laboratory or in an adjacent area. These studies are 'semi-attended', with a knowledgeable sleep-laboratory technologist nearby if technical or patient problems develop.

Future Role of the Sleep-Laboratory Technologist

The sleep-laboratory technologist's role in determining the appropriate CPAP or bi-level pressure therapy level cannot be overemphasized. The technologist conducts the study, usually independent of direct oversight or physician input. Physician review of each study is mandatory but is always retrospective. Accordingly, physician trust of the sleep-laboratory technologist and respect for his or her judgment during the sleep study is paramount.

Although each sleep laboratory has general guidelines about how therapy is to be titrated, these do not substitute for clinical judgment and experience and should not be used to prevent experienced technologists from applying their clinical knowledge of sleep-disordered breathing and positive pressure therapy to optimize patient acceptance and use of this therapy. Indeed, because little uniformity exists among sleep laboratories about CPAP titration and standard protocols outlining the optimal way to titrate CPAP therapy do not exist, the value of a knowledgeable technologist becomes even greater.

Patients may benefit considerably by having a technologist knowledgeable about CPAP available to educate them about this type of therapy. Mask choice and the finding of the best interface for a patient may be optimized by this approach. One concern raised by the increasing use of home-based CPAP therapy is the lack of a technologist present to educate the patient about CPAP therapy and to obtain the best-fitting CPAP mask possible. It seems obvious that equipment and therapy education can be done at other times. However, this important aspect of the conventional sleep laboratory should not be overlooked as auto-CPAP use becomes more widespread. The role of the sleep-laboratory technologist in optimizing clinical outcomes has not been closely studied but may be extremely important in obtaining the best result possible for patients using CPAP.

The technologist is in an ideal position to become a co-manager with a qualified physician of patients with sleep disorders, particularly those patients with OSA, because of the necessity for ongoing use of CPAP equipment. Technologists usually have the knowledge necessary to assess patients who have an established diagnosis of OSA who are on therapy. Troubleshooting mask or pressure complications, assessing subjective response to therapy, and providing ongoing patient education are all within the reasonable scope of practice of a sleep-laboratory technologist.

Managed care organizations may find a partnership between sleep-disorders clinician and sleep technologist attractive. The sleep technologist serves not only to conduct sleep studies but also develops an expanded role as a physician extender. This model can be advantageous for all parties involved. The technologist's role is enhanced by this additional role; the physician and health care system become more productive as more patients are cared for in a more comprehensive manner by the same number of staff. Cost-effectiveness and patient satisfaction are key goals for all health care systems. Although data showing benefits to such a program are not available, it is likely that sleep-laboratory technologists would be cost effective in this additional role. Patient satisfaction would also likely increase because a knowledgeable professional is readily available to help the patient receive optimal benefit from CPAP or bi-level PAP therapy.

In Conclusion

Treatment of OSA has rapidly evolved over the past two decades from lifelong surgical tracheostomy to positive airway pressure therapy. CPAP and other positive airway pressure therapies are now the mainstay of OSA therapy. Striking advancements in positive airway pressure technology and increased clinical recognition of these disorders have made this evolution possible.

As the number of therapeutic options has increased, finding the best therapy for any patient has become more complicated. Fixed level CPAP is the most commonly prescribed therapy. Bi-level PAP has a role in the treatment of selected patients but at a higher cost. The role for auto-titrating CPAP will likely increase, especially if the potential cost savings, theoretically possible with such devices, are realized. Furthermore, if manufacturers of auto-CPAP units sell these devices at a cost to the health care consumer at or near standard CPAP costs, their use in the treatment of OSA may increase dramatically.

As the field of sleep disorders medicine matures as a clinical endeavor, the role of the sleep disorders laboratory and its personnel will also change. Sleep-laboratory technologists could play a larger role in ongoing patient management as physician extenders and care coordinators. What seems clear is that patients with sleep disorders are being identified commonly, that effective treatment for many of these disorders (especially sleep-disordered breathing) is available, and that technology aimed at sleep-disordered breathing is rapidly advancing toward systems that require less moment-to-moment technical supervision. The need to manage the growing number of patients evaluated and treated for sleep apnea, in cost-conscious medical systems, compels new approaches to the diagnosis, treatment, and long-term management of these patients. Physicians, sleep-laboratory technologists, the medical equipment industry, and health care administrators each have important roles in developing optimal approaches to the management of this important and growing group of patients.

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Discussion

Chediak: I have a little experience with the various self-adjusting nasal positive airway pressure devices commercially available now. Although my experience is small, it's been enough to have me concerned about using them for unattended clinical studies. I want to point out that the published clinical studies on self-adjusting positive airway pressure devices have compared automatic laboratory titration to manual laboratory titration, not manual laboratory to automatic home titration. In my personal experience in the laboratory, the self-adjusting devices don't work well. I recognized that they overreacted or underreacted under a variety of situations that left me wondering how they would behave in the home setting if there was no one there to conclude that the machine is inadequately adjusting pressure. I've tried on about 7-8 occasions to allow a machine, an autotitrating machine, to go through the night by itself to save my technician time. My technician has yet to allow a machine to go through the night in the automatic mode. Inappropriate automatic adjustments made the technical staff attend to the patient at least as often as with manual titration. This effect occurred with the Autoset, the Virtuoso, and the D-Pap units. The devices that respond to vibration have responded to cough and speech because cough and vocalization are, after all, a form of vibration. The ones that respond to flow limitation are fooled by leaks. From my personal experience in the laboratory, I would be hesitant to use them at home in an unattended fashion, unless you have a situation where monitoring in an attended setting is not available.

Minkley: I want to comment on your protocol for titrating CPAP. One issue—I think you addressed it a little bit—is you always go 'up' and not just come down. When you are titrating to arousals, there becomes some rhythmicity to the arousals if they are associated with upper airways resistance syndrome. However, when arousals are seen that are not cyclic or rhythmic, oftentimes that is a sign of intolerance in the patient. Either slowing down the titration or actually backing down sometimes will solve that problem, and then increasing by smaller increments as you suggested. That's titration of the ideal patient—where everything works, and as we know, those only appear at conferences like this. In the sleep center, we often have more severely affected patients who have multiple problems. So, we're also dealing with beginning with continuous positive airway pressure and then switching to some mode of bi-level PAP ventilation, then also adding in oxygen at the same time. It's difficult as a technologist to pull back and continually look at what is our role. Our role is to give you, the clinician, as much data as possible, to make some decision on what therapy you will order. Often that decision will have to be made not on being able to order the optimal, perfect therapy, but some compromise. It's important that we be able to give you the information you need in a time frame to make that decision.

Atwood: Certainly this titration is not meant to be 'the truth' about how to do CPAP. There are a million different variables that come into play in each and every patient, simultaneously, but to the extent that we can standardize at least a general approach to this, I think it would be desirable. The other thing I really didn't touch on is When do people in sleep laboratories switch to bi-level therapy? Some laboratories will say "Anything > 10 cm CPAP, we don't think the patient's going to tolerate this." We think compliance will be poorer, although certainly we've published, at least in abstract form,[1] data that even at much higher levels of CPAP there was no difference between compliance at several months with lower levels of pressure. But, some labs have an arbitrary level at which they switch over to bi-level therapy. In our facility, we don't. We generally go by patient tolerance and how well they accept the CPAP. I'd like to hear other people's thoughts.

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Minkley: I think the important thing that you address is that there does need to be a protocol, and frequently there is not. If more of us had protocols and could report the results that would help in the development of therapeutic decision trees.

Atwood: It certainly helps people make sense out of what goes on at other people's facilities.

Barnes: If you're looking for partnering, I know that respiratory therapists qualify. They are experts at weaning patients from mechanical ventilators and adjusting mechanical ventilation. They have experience with analysis of volume, flow and pressure wave forms, and hemodynamic monitoring. Therefore, to add additional monitoring parameters and assist with adjustments in CPAP and bi-level pressure ventilation would be a natural for respiratory therapists.

Strohl: How often are you successful in titrating CPAP? Is it different in a split-night or a whole-night titration?

Atwood: Studies have shown there's a 60-80% success rate with initial CPAP.

Strohl: I mean with a titration night. You're pleased and the technician is pleased that you got the right pressure.

Atwood: I think in our facility it's about 80%, taking all comers.

Strohl: So, 1 out of 5 titrations is not acceptable.

Atwood: That's a reasonable ball park figure, yeah.

Strohl: That's interesting. And is it the same, do you think, for split night as well as whole night?

Atwood: I think split nights are more problematic, and there is a higher number of what we would consider unfinished titrations.

Strohl: What do you do if it's unfinished?

Atwood: I will frequently empirically increase the pressure a little bit, and then see how they do. Sometimes I bring them back to the lab for another night, and that shows my, perhaps, practical bent more than my theoretical bent.

Strollo: Our protocol with titration is that we go up 1 cm, and if we have arousals we start going down.

Estes: Just to give you some rough breakdown of practice in the States today, if you look at what kind of modality is being put on patients, CPAP constitutes about 85% of the patients on therapy, with bi-level therapy making 10%, but 5% (up from roughly < 0.5% a year ago) are in the autotitrating category. That's patients going home. I submit that a lot of the discussion we had on the previous topic plus this very recent discussion about how often are you successful in titrating, that's the niche that the auto machines are filling. Pressure needs do change day-to-day, so taking that snapshot—one night—could be what drives our compliance issues. How do we know that needs change every day? Download an auto machine and take a look. There have been several studies.[1,2]

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Strohl: How do you know that's going to affect compliance?

Estes: Burk et al presented a study at the APSS[1] [Association of Professional Sleep Societies] where they took CPAP patients who were fairly compliant and actually improved their compliance by putting them on an auto device. It was a fairly well-designed study.

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Strollo: Those were small changes, though. Those were people who were compliant, and the hours did go up modestly, but the role for autotitrating devices, I think, maybe if we had a little more time at the end, maybe we'll adjourn down to the pool and talk about this some more.

SEE PAGE 342

For a listing of sleep organizations in America

Positive Pressure Therapy: Compliance and Partnering with Home Care

Karen A Studnicki CRTT RPSGT

Introduction
Optimizing the Laboratory Experience
Evaluation & Titration of Therapy
Acceptance of CPAP at Time of Prescription
Initial Follow-Up Strategies
Monitoring Compliance Long Term

Introduction

Successful implementation of positive pressure therapy requires coordination among the patient, the sleep laboratory staff, and the home care provider. Effective interactions augment patient compliance and avoid the pitfalls and barriers to optimal patient care. This discussion highlights the need for on-going patient education with experienced and qualified sleep technologists in order to shepherd the patient through the testing procedure and the transition from a sleep laboratory trial of positive pressure therapy to the continued use of therapy in the home.

Over the years, a number of studies have provided conflicting information regarding the value of patient education programs, careful phone follow-up, return visits to the clinic, and home visits by the home care companies in augmenting long-term compliance with continuous positive airway pressure (CPAP).[1-5] CPAP does appear to be reasonably well accepted by patients[6] and most agree that patient education and monitoring protocols favorably influence CPAP acceptance and compliance.

Krieger et al[1] suggest that optimal treatment with positive pressure involves three components: (1) a proper CPAP titration and prescription; (2) patient willingness to be set up with CPAP (ie, acceptance), and (3) patient commitment to continued use (ie, compliance).

In our Sleep Center, we have found that the respiratory care professional (RCP) can be a valuable asset to the patient, the sleep laboratory, and the home care provider. The RCP can serve an essential role in optimizing patient acceptance and compliance through prestudy patient education as well as during the positive pressure titration procedure, including evaluation and resolution of potential problems with CPAP use or equipment. This facilitates a positive patient experience during his or her initial exposure to this therapeutic modality. Follow-up intervention and involvement in support outside the laboratory environment provide both patient and family with a familiarity and continuity of care that may be beneficial to long-term compliance.[7-9]

Fletcher and Luckett[2] have reported three factors that could affect compliance with nasal CPAP; (1) motivation, which is tied to severity of symptoms and satisfaction with mode of therapy, (2) perceived complications of CPAP use and, (3) degree of education regarding pathophysiology of OSA and importance of treating it.

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Optimizing the Laboratory Experience

In my experience, patient education, provided prior to the actual positive pressure titration procedure is a key factor in the acceptance of CPAP therapy. At the University of Pittsburgh Medical Center, during the initial physician interview, the causes and consequences of sleep apnea and the various therapeutic modalities used to treat the disorder are discussed with the patient. Although surgical options are presented, the emphasis is usually on positive pressure therapy as the initial intervention. The patient receives a brochure distributed by the American Sleep Apnea Association (ASAA), which also provides detailed and informative discussions on sleep apnea. If needed, patients are then sent to the Pulmonary Sleep Evaluation Center to be scheduled for overnight polysomnography.

At this time, the patient is provided with the opportunity to tour the laboratory and ask questions of the technologist regarding diagnostic testing and is briefed regarding what to expect. The technologist reviews how the test is conducted and explains the procedures and equipment used during testing, and may provide the patient with an opportunity to try breathing on the device under the guidelines of the laboratory. Every patient is given the opportunity to view several educational videos provided by the ASAA and, if time is limited, the videos are shown on the night of testing. Often, at the clinic visit, the patient is accompanied by a spouse or bed partner, and both the patient and the family can be counseled on the beneficial effects of CPAP. We recognize that family support may be crucial in facilitating the patient's acceptance of therapy.

Evaluation & Titration of Therapy

Prior to the patient's therapeutic trial of positive pressure therapy in the laboratory, the technologist determines the optimal mask interface with regard to fit and comfort. Although Pepin et al[10] found that compliance remains high in spite of patients' experiencing side effects related to CPAP application, other data on compliance suggest that side effects[11] including problems with mask adjustment and discomfort are some of the biggest obstacles to acceptance and compliance.[12,13]

Many practitioners believe proper mask fit is crucial to CPAP compliance. We have found the most common complaints to be skin irritation at the bridge of the nose and mask leaks. Familiarity with and knowledge of the availability of the many types of interfaces is important to deciding what might work best for an individual patient and may optimize the positive pressure titration in the laboratory. In our laboratory, before they go to sleep, patients choose the interface they prefer by breathing through both nasal masks and pillows until they are comfortable. By optimizing mask fit prior to the start of the study, less 'fine tuning' is required during follow-up outside the laboratory.

During the positive pressure titration procedure, it is important for the technologist to determine and evaluate the most troublesome patient concerns and problems. Carefully written patient observations during the study provide the reviewing physician with information essential for determining the optimal pressure and interface for the positive pressure prescription. The technologist is able to supply the physician with information regarding problems encountered during the night, from pressure intolerance or difficulty sleeping to the advisability of incorporating a ramp or humidifier to help the patient utilize therapy at home. Morning questionnaires completed by the patient are also helpful in evaluating the night in the laboratory and for providing insight into whether positive pressure therapy will be accepted.

Upon completion of the therapeutic night in the laboratory, the most commonly asked question of the technologist in the morning is What happens now? The importance of using CPAP is emphasized by the technologist, and the technologist becomes an important part of the transition from the trial of positive pressure therapy in the laboratory to home use.

Acceptance of CPAP at Time of Prescription

Careful coordination among the patient, the home care company, and the technologist is essential to facilitate patient compliance. Prior to leaving the laboratory after the study, patients understand that if they elect positive pressure therapy, they will need to choose a home care company. In our sleep center, studies are reviewed by the physician the following morning, and patients are usually notified of the results within 24 hours. The patient already may have made his or her choice of home care companies (often on the basis of proximity to home). In some instances, patients may be unfamiliar with how home care works or do not have the knowledge of where and how to begin. The RCP can assist the patient by explaining how to examine cost and insurance coverage information and in communicating to the home care provider the specifications for the required interface and positive pressure device. In our laboratory, we often contact several home care companies to explain what is needed and have them contact the patient to discuss therapy set up and insurance issues. In the managed care environment, specific home care companies are mandated. The sleep laboratory staff should be aware of those companies participating in health maintenance organizations and be familiar with the necessary steps in providing therapy to this group of patients.

Much of the data published on patient compliance indicate that the first few weeks are critical in reinforcing commitment to use.[1,3,4] It may be assumed that the sooner the patient is set up at home, the better the compliance will be. When the technologist calls with the physician prescription information to the home care provider or durable medical equipment supplier (DME) of choice, the patient usually receives the unit within 24-48 hours.

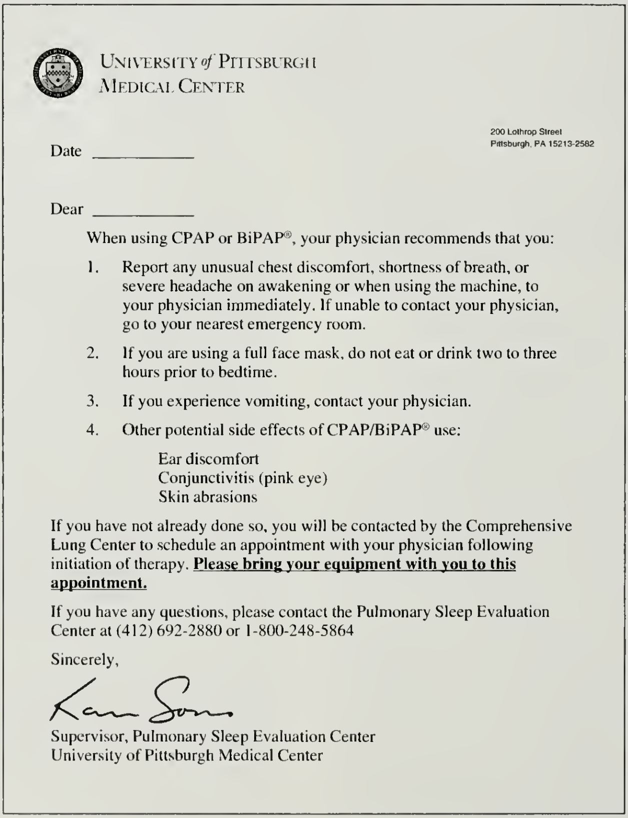
In those patients in managed care programs, the prescription information must be forwarded to the primary care physician. It has been our experience, that these patients often may be at risk for delays in implementation of therapy. As part of quality assurance, we track the progress of all patients and assist patients in expediting therapy set up at home.

Once patients initiate therapy through a home care provider, we mail the patient a brochure on sleep apnea and a letter developed by our laboratory (Fig. 1) explaining some of the complications that may be encountered with positive pressure therapy, such as unusual chest discomfort, shortness of breath, or severe headache on awakening, and to contact the physician immediately should these occur. It also lists other potential side effects as ear discomfort, conjunctivitis, and skin abrasions. Those patients for whom an oronasal mask is prescribed are counseled not to eat or drink 2-3 hours prior to bedtime. A.W.A.K.E. (Alert, Well and Keeping Energetic) and American Sleep Apnea Association applications are made available to the patient. The A.W.A.K.E. group meets regularly and addresses issues of compliance, among other topics, and is fully supported by most home care companies.

All patient information is entered into a database from which we can easily access prescription information, patient's choice of DME, set up date, and the most current hour-meter readings. The database is updated as information is provided to us from the home care companies. Compliance data can also be easily extracted from such a database.

Initial Follow-Up Strategies

Patients are contacted by telephone by the staff approximately 2 weeks after initiation of therapy to discuss any prob-



UNIVERSITY of PITTSBURGH
MEDICAL CENTER

200 Lothrop Street
Pittsburgh, PA 15213-2582

Date ___________

Dear ___________

When using CPAP or BiPAP®, your physician recommends that you:

1. Report any unusual chest discomfort, shortness of breath, or severe headache on awakening or when using the machine, to your physician immediately. If unable to contact your physician, go to your nearest emergency room.
2. If you are using a full face mask, do not eat or drink two to three hours prior to bedtime.
3. If you experience vomiting, contact your physician.
4. Other potential side effects of CPAP/BiPAP® use:

   Ear discomfort
   Conjunctivitis (pink eye)
   Skin abrasions

If you have not already done so, you will be contacted by the Comprehensive Lung Center to schedule an appointment with your physician following initiation of therapy. **Please bring your equipment with you to this appointment.**

If you have any questions, please contact the Pulmonary Sleep Evaluation Center at (412) 692-2880 or 1-800-248-5864

Sincerely,

Supervisor, Pulmonary Sleep Evaluation Center
University of Pittsburgh Medical Center

Fig. 1. Sample follow-up letter for patients who are being treated with continuous positive airway pressure or bi-level positive pressure.

lems or concerns with the therapy and/or equipment. These are addressed at once and can include changing the interface, adding a ramp, providing humidification, or modifying the current therapy. A written follow-up report is then placed in the medical record, which provides the physician with a follow-up assessment on the patient's return visit.

At the University of Pittsburgh Medical Center, we provide all patients with a letter that explains the need for a return visit to the clinic once therapy is initiated at home. In this letter, we ask that the patient bring the CPAP or bi-level unit to the follow-up clinic visit. At this time, the importance of regular CPAP use is re-emphasized by the physician. During the visit, we are able to inspect the machine, verify the positive pressure prescription, and obtain an objective meter reading from the positive pressure device.

The relationship between the home care provider and the sleep laboratory is an important one. It is essential for the sleep center to be involved in establishing guidelines and standards of practice with the home care provider. This should include having a qualified RCP set up the therapy in the home, with orientation of the patient to the unit and instructions on cleaning and light maintenance of the equipment. Telephone follow-up within 24 hours should be done by the DME, although we do this as well in selected cases to establish and address any first night issues regarding positive pressure use. A monthly hour-meter reading, an objective reading displaying number of hours used, should be reported the first few months to assess patient compliance. We require that brief summary reports of any mask problems, irritation, or other concerns also be forwarded to the sleep laboratory.

Many of the home care companies we deal with on a regular basis call in often to consult with the technologists concerning what can best be done to optimize individual patient care, especially in those patients who have difficulty initially adjusting to therapy. There is often an exchange of information regarding how the patient reacted in the laboratory as compared to how he or she is handling therapy in the home. Occasionally, a conference call with the patient, home care provider, and sleep laboratory provides a useful means of problem solving and providing support.

When the patient returns to the clinic for the follow-up appointment, these initial home care reports are available in the chart for physician review. At this time, the sleep laboratory can be revisited as needed, to re-size masks, try other interfaces, or discuss ways to prevent leaks around the mask. Often, the patient is able to talk with the same sleep technologist who performed his or her test and is familiar with the patient's situation.

Monitoring Compliance Long Term

Much of the information on compliance suggests that there are no ideal ways to objectively monitor these data. Some of the more recent CPAP units objectively record time at pressure so that the patient cannot 'cheat' and run the unit as if it were in use every night, though the data suggest this happens rarely. There are software programs available that provide charts and graphs with useful compliance information.

The key issue is not how to measure compliance in a more 'tamper proof' way—although this certainly provides important information, especially because it has been reported that patients often overestimate hourly use via questionnaires—but how to improve patient acceptance of therapy. Technologic advances relative to mask comfort and less conspicuous and quieter positive pressure units can possibly positively influence patient acceptance. Rolfe et al[13] suggest that improvement in CPAP design and careful patient training may improve acceptability of CPAP substantially. Kribbs et al[14] also suggest that CPAP use might be enhanced if more emphasis were given to efforts designed to reduce fear of and intrusiveness of therapy.

The final commitment is ultimately on the part of the patient. Patients need to be made aware of the beneficial effects of CPAP. It is the role of the respiratory care technologist to educate and support the patient in establishing that awareness and to identify those factors that can favorably affect patient compliance with positive pressure therapy.

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Discussion

James: The things that you mentioned are important. I challenge you and everyone in this room to take that one step farther. Obviously, all the things you talked about are labor-intensive, not only for the sleep lab and for the physician, but also for the home care company that gets involved. To show that those things are beneficial—which we intuitively know—we need to go a step farther and show the outcomes. Only when you monitor those data to make sure they are objective do you have a way to provide the outcomes information back to the payer that can prove its feasibility.

Chediak: I always wondered about the need to be so rigorous with CPAP compliance. My queries stem from a couple of indirect pieces of evidence. One, the covert monitoring study by Kribbs[1] demonstrated to us that CPAP use is, at best, 4 hours out of the night and 5 out of 7 nights in half the CPAP users. That's at best. So, that's half the night in half the people. Yet He's retrospective study looking at survival in OSA[2] showed that CPAP users live longer than non-CPAP users. You wonder, "Well, did they have good compliance?" They probably didn't. I can't imagine them being any better than the covert monitoring study, where participants were being followed more closely and getting periodic office visits. If one examines other studies that looked at end points, such as sleepiness and so forth, very few of them have gone out of their way to check the compliance on CPAP.[3] Yet they demonstrate benefits in blood pressure control and decreased sleepiness. I assume that CPAP compliance in those patients is no better than half of them using the device 4 hours a night. So, does CPAP compliance really make that much difference? Or maybe I'm just being a little bit lazy because I can't get the compliance in my own patients to be any better, and I want an excuse for not having to fight them so hard. Perhaps if they used it more, they would be even better. Maybe they would live to be 120 and their blood pressure would be 60/40 or something. I don't know. The point is that compliance with CPAP is poor. Compliance was likely no better, one would intuitively think, in those studies that have demonstrated benefit in outcomes like survival and sleepiness. Yet we're fussing over giving the perfect titration, using it every night, using it 8 hours, whenever you sleep, during naps. Perhaps we should be less concerned with compliance at least in patients with self-reported and witness-corroborated resolution of symptoms.

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Strollo: I think we're brutally honest in the sleep world in terms of whether we're making a difference. We've been asking these questions: the proper way of testing. If you look at compliance with *any* medical therapy, be it hypertensive medications, which are a lot easier to take, or asthma medications, compliance is 50%. One of the questions that you raise, Alex (Chediak), is How much time does a patient have to be on a CPAP machine to receive benefit? Does it have to be the entire night? Intuitively, we would think it *should* be the entire night. Will auto-set machines improve the time that the patient's on the entire night? I think the jury is out on that right now, but that's one of the hopes with regard to auto-set machines or autotitrating machines. We've really worked hard over the past few years to set up the follow-up clinic, and it kills us clinically. There's no question about it. It's very time intensive, and we've had the luxury of doing that. I don't know whether the current economic constraints are going to allow us to continue. We're now looking to move to extenders, and we're hiring a nurse practitioner to offload it. It's clearly not cost-effective to have a physician do it. I'd love to have a respiratory care provider do it. The problem is you can't get payment for a respiratory care provider administering this kind of care. Respiratory care providers would be excellent people. One of the ways that we use compliance is that when people clearly aren't using their machines when they come back, we withdraw the machine much like the Edinburgh group does. I think that if you're going to look at these monitors, you have to be prepared to take the machine away, and if the patient's condition is severe, shunt them into alternative therapies. We're going to talk a little more about that tomorrow, but therapy that's 50% effective or 70% effective no matter how you judge it is better than 0% effective if the CPAP machine is in the closet. I think that's the practical aspect of compliance. I think we can improve the number of hours, but whether that really improves clinical outcome, I don't know.

Brooks: More important than the hours is the clinical outcome. If the patient says "I'm not sleepy anymore. My wife has moved back into the bedroom, and I'm doing a better job at work, and I'm not falling asleep at the water cooler anymore," who cares how many hours he's using the machine? He's better.

Strollo: I agree with you. I don't touch those people if they're doing well and their blood pressure is well-controlled, but in the patient who frequently comes back and says "I'm still sleepy." And then we look at their hour meters, and they have an hour and a half a night on their hour meter, you don't need to do an MSLT. You don't need to do another CPAP titration. We say, "Here are the data. You're not using your CPAP. The option is to increase your hours or to seek other treatment." So that's another way that we find objective compliance to be useful in our clinical management that Karen (Studnicki) didn't touch on.

Minkley: This has been a struggle as long as I've been involved in sleep. How do we determine compliance and in the studies that are out there what did they use as a marker? It's very variable. So, when they report compliance, it depends on how they define compliance, as you mentioned. Was it 5 hours a night? But then the patient who only sleeps 4 hours would be noncompliant, although they used it 100% of the time. There are those issues from our discussion previously about how to select patients who need to be studied in sleep and looking at their symptomotology and their presentation. There doesn't seem to be any good agreement on how to select patients. So, it's no wonder we can't come to any agreement on how to determine whether a patient is compliant. We haven't even clearly defined who needs to be studied and what markers there are, so what would we use as a marker for whether a patient is compliant or their symptoms are resolved? Many clinicians who follow these patients may not have had the education to clearly define therapeutic goals, so have no measure for resolution.

Strohl: I think you have to articulate your goal of therapy to the patient, then to the referring physician. If your goal is to eliminate snoring, that's fine. If your goal of therapy is to reduce sleepiness, that's fine. So, I think you have to identify for the patient what your goals are and how you are going to reach them, and then use objective compliance as well as subjective "how do I use it?" in a pattern of assessing the patient more closely than by Medicare guidelines, which is once a month. They have to be checked after 1 month of therapy—like the traditional oxygen guidelines. You work with them in the first week and make sure that they know what they're doing. You reinforce education. You do those things. I think that's how you optimize the chance of treatment with CPAP. You're not going to get everybody, but that's how you optimize.

Woodson: My office is kind of at the end of the hallway. I get sent those people who have had a problem with CPAP. I see 3 types of people fail CPAP. The first group told you when they first walked into the office that they weren't going to wear the mask, and they come back and they say "Doc, I'm not going to wear the mask." OK. And I found that generally all the education in the world and all the time generally has little effect on this group of people. In the second group, are those patients who are claustrophobic, have some serious mask-intolerance problem, or some other medical issue This is a very recalcitrant group. Third, I see people who have had very poor education—people who have not had the therapy very well explained to them. They come in with a misperception as to the treatment. They think that somewhere out there there's an easy treatment. They come into my office to fulfill that expectation. I think it behooves all of us in sleep medicine to educate our patients and let them know that no form of treatment is easy, at least for a large number of people. Education is extraordinarily important, both before you initiate treatment, before you get the sleep study, and then, certainly, on an ongoing basis. We have to press the third-party carriers about how critical that step is. You need to spend the money on just the simple things of actually dealing with the patients face to face.

Barnes: The desaturation that occurs during OSA is a serious problem. During this conference, I have heard comments about sleepiness and the quality of life. How often do people with serious obstruction die in their sleep? Do you attempt to identify the group with serious desaturations and monitor their compliance to CPAP protocols?

Strollo: I think that the discussion got a little clouded this morning from the standpoint that we don't know how to identify patients who have severe disease. I think that most people in sleep medicine can identify people who have severe obstruction and severe nocturnal desaturation and need to be treated. That's not where the clinical conundrum is. The clinical conundrum exists in these people who have a mildly elevated apnea-hypopnea index and complain of fatigue or mild daytime sleepiness and have some degree of insufficient sleep. Where that becomes a disease is a question. I think all of us push hard in folks who have severe nocturnal desaturation, nocturnal angina, nocturnal dysrhythmias. Treatment has to be done in an expeditious fashion and the patient has to be objectively compliant. If not, there still is a role for tracheostomy in 1997. I clearly don't do tracheotomies on a large majority of our patients, but there's a subset of folks who still get a tracheostomy.

Minkley: One of the requirements for sleep-center accreditation is that some attempts be made to make sure that patients are followed up—treatment was initiated if treatment was ordered—and for patients who are negative in a study there is some degree of follow-up when they go back to their referring physician—to make sure that the symptoms are dealt with, maybe in another manner. So, for those patients who come through a center, particularly centers looking toward accreditation, there is a mandate that there be some degree of follow-up.

Chediak: I just want to tell a little story that I think fits right in with compliance issues. We're all blaming the patient for lack of CPAP use. However, even physicians can be responsible. Last Friday, I had a sleep apnea patient who also had complete bilateral nasal obstruction and who was poorly funded. As soon as I completed the interview and exam, I put him in my sleep lab during the day and let him sleep there for about 4 hours. We let him sleep for an hour, documented he had severe sleep apnea and desaturations, and then we tried nasal-oral CPAP, thinking maybe we could get the pressure through his mouth. However, nasal-oral CPAP failed, and he was severely incapacitated with excessive daytime sleepiness. Further, he could not sustain sleep because dyspnea and choking regularly awakened him. He would drive around Miami at night trying to stay awake until such time as he just couldn't resist sleep, and then he'd pull into the nearest parking lot he could find and sleep in the car. He'd been arrested several times for this behavior. So, I said "You need a tracheostomy," and I called my fellows, and I said, "I'm going to send this person to the emergency room so you can treat him with a tracheostomy. He has bi-lateral nasal obstruction and nasal CPAP has failed. Please admit him and have the ENT specialist see him over the weekend." I called his internist and advised him similarly. So, he was admitted Friday. He first went home to think about it, and then when he couldn't sleep, he came back at about 2:00 in the morning. Unfortunately, he was admitted by a house officer to whom I had not spoken, who put him on oxygen and in the ICU and then tried nasal CPAP. On Monday morning on attending rounds one of my other colleagues, a more experienced pulmonary physician, (but not necessarily in sleep apnea) saw him, and since the patient admittedly felt better sent him home on nasal CPAP. There's a certain amount of physician lack of understanding of CPAP use, even within educated groups. The obvious—the patient who can't breathe through his nose—was missed. In this case, CPAP is doomed to fail and lack of physician recognition of the difficulties with NCPAP, not poor patient acceptance, is the true cause of CPAP nonuse. Fortunately, I was able to rectify the situation.

James: I can't resist commenting on the affordability of self-titrating CPAP devices. There are some out now that are just pennies more than a standard CPAP.

Strohl: There are some people who are really, truly claustrophobic. There are some people to whom you show this machine and that's not what they want. The other group that Ira Rubenstein would talk about is those who have nasal inflammation. You put CPAP to them and they have rhinitis or sinusitis, and it feels like an ice pick went through their head. I wonder if pretest, pre-CPAP, you could start to predict those. Do you have any feel on prediction of success even before they even use it in the sleep laboratory or at home? Any strategies?

Studnicki: Some patients come into the lab, and even before they are scheduled for an overnight study, we give them a tour. We discuss with them their options, eg, positive pressure therapy, and their first response is "No way." You have a pretty good feeling that you're going to have to really work with those patients to try to get them to "play ball" with CPAP.

Strohl: How often are you successful with that group of people?

Studnicki: I don't know percentage-wise. We've been …

Strohl: How about more often than not, or something like that? Or less often?

Strollo: I'll make a couple of comments about it because it involves our laboratory. My partner 'never says die' in terms of CPAP. I would say that in difficult patients, clearly, you don't get 100% compliance in that group. There are some patients you salvage, but it's quite low. And, as Karen (Studnicki) and some of the other folks commented, although you *can* identify people who are going to be trouble in the lab, I know of no study that's actually looked at compliance in the context of looking at it up front in terms of saying "I won't accept therapy." Most of them have examined compliance sort of post-hoc.

James: I would like to add something controversial, since that's the spirit of this meeting. If you're looking for an in-office test to assess whether a patient might accept CPAP, there was actually some work done by a neurologist named Govindan in West Virginia.[1-3] He did intracranial blood flow studies on patients and found that those who didn't tolerate CPAP—it wasn't a psychological reason, they just for some reason couldn't tolerate it—had changes in intracranial blood flow on CPAP. That's done with Doppler and with thermography.

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Brooks: Sometimes I think it tends to go more the other way in kids, where I will recommend surgery, and the family is absolutely against it. If we're in the spirit of anecdotes, I had a patient with a mucopolysaccharoidosis, which, if you remember, involves a facial deformity, small jaw—bad anatomy. The face itself has a broad sunken bridge of the nose, and the children are retarded. I thought that there was no chance of our finding a CPAP mask that would (a) fit and (b) this kid would tolerate, because he wouldn't even allow us to give him aerosols to treat his asthma. But the family was insisting on a CPAP trial—so we'll give them a CPAP trial and see what happens.

Strohl: In terms of the things that I do, I have a CPAP machine in the office where I examine them, and I first put it on their hands so that they can feel its pressure, and I then put it on their nose. So far, about 10% of the people have a "startle" response to CPAP, especially if I don't explain exactly what that machine is. But if it's sitting up there on the counter, one of the things is just seeing it as part of the examination. If there is one of the machines in the office and we give an explanation of surgery, we can start to better explain what options there are, and make a decision with the patient on how to treat them. One of the best things I have found is if they can't breathe through their nose with CPAP, I examine more closely the nasopharynx or give nasal steroids before a sleep study. So, I mean you can do some simple tests in the office with just the machine there, without it being 10:00 at night while they're putting on the electrodes.

Minkley: These patients are seeing Dr Strohl ahead of time, but unfortunately, what I'm hearing as I travel around the country and talk with technologists who work in sleep centers is that more and more and more sleep centers are now having to accept direct referral patients. More and more patients don't have the advantage of having the education like you give your patients or of seeing CPAP instrumentation prior to their visit. In our own center, as the structure of the center has changed from a clinic/sleep center base to separate facilities, we have had to become more aggressive in our education because it, in many cases, is the patient's first introduction. When the patients admit to or show signs of claustrophobia with the mask, we're finding that a number of those, and I'm hearing this from other sleep technologists, too, do tolerate the full face masks. That seems counterintuitive, but they do. At our institution, some people report the use of hypnosis with these patients and some degree of success. So, again, persistence seems to be the name of the game, and going back again to what we talked about is who is case managing these patients, and who is setting up clinical and treatment goals, and who is following them? Certainly in Karen's (Studnicki) case, it is that center, but I'm not sure that that is the norm across the country. The more dispersed the diagnostic testing becomes, the more I have a concern about who is going to take the reins to case manage these people and make sure the treatment plan is actually implemented and the goals are reached.

Strollo: I think our center is somewhat unique. It represents in many respects a 'best-case scenario' in terms of controlling for a number of these things. Now, is that cost-effective? We haven't compared it to community management. It seems almost intuitive, but … the gauntlet is down, certainly, to prove that. I want to revisit some of the issues regarding autotitrating devices. In autotitrating devices, where 'value added' frequently is obtained, is allowing the titration to occur in a rational manner. One of the things that I've been struck by when I've gone around the country and visited other sleep centers, is the way titrations are done. Frequently pressure and flow are not placed on the polygraph tracing, either the computer or the paper. Our laboratory also is unique from the standpoint that all our technicians are respiratory therapists who are very familiar with wave forms and are keyed into that. But, unfortunately, that's not the case in some centers that don't look at flow patterns. An autotitrating device may be substantially better in terms of executing a titration than a center that doesn't look at those parameters. No one has really studied that in any formal fashion that I'm aware of.

SEE PAGE 342
For a listing of sleep organizations in America

CORRECTION

In the paper Volsko TA. A Pediatric Asthma Clinic Pilot Program Reduces Emergency Department Visits, Hospitalizations, and Cost of Care. Respir Care 1998;43(2):107-113, Appendix 2 is inaccurate. The correct version of the Asthma Action Plan is printed below. We regret the error.

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Peak Flow Range :	
PLAN:	
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Peak Flow Range :	
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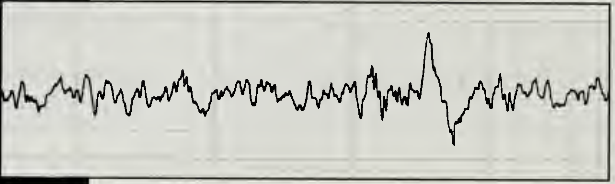

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American Association for Respiratory Care

MEMBERSHIP APPLICATION

Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

- ☐ Active
- Associate
  - ☐ Foreign
  - ☐ Physician
  - ☐ Industrial
- ☐ Special
- ☐ Student

Last Name ______________________________

First Name ______________________________

Social Security No. ______________________________

Home Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __—__ __ __ __

Phone No. (______) ______________________________

Primary Job Responsibility (check one only)

- ☐ Technical Director
- ☐ Assistant Technical Director
- ☐ Pulmonary Function Specialist
- ☐ Instructor/Educator
- ☐ Supervisor
- ☐ Staff Therapist
- ☐ Staff Technician
- ☐ Rehabilitation/Home Care
- ☐ Medical Director
- ☐ Sales
- ☐ Student
- ☐ Other, specify ______________________

Type of Business

- ☐ Hospital
- ☐ Skilled Nursing Facility
- ☐ DME/HME
- ☐ Home Health Agency
- ☐ Educational Institution
- ☐ Manufacturer or supplier
- ☐ Other, specify ______________________

Date of Birth (optional) ____________ Sex (optional) ____________

U.S. Citizen? _____ Yes _____ No

Have you ever been a member of the AARC? ______________________

If so, when? From ______________ to ______________

Preferred mailing address: ☐ Home ☐ Business

For office use only

FOR ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment ______________________________

Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __-__ __ __ __

Phone No. (______) ______________________________

Medical Director/Medical Sponsor ______________________________

FOR ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment ______________________________

Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __-__ __ __ __

Phone No. (______) ______________________________

FOR STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

School/RC Program ______________________________

Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __-__ __ __ __

Phone No. (______) ______________________________

Length of program

- ☐ 1 year
- ☐ 2 years
- ☐ 4 years
- ☐ Other, specify ____________

Expected Date of Graduation *(REQUIRED INFORMATION)*

Month ________ Year ________

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 484-2720

American Association for Respiratory Care **MEMBERSHIP APPLICATION**

Demographic Questions

We request that you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned

☐ High School
☐ RC Graduate Technician
☐ Associate Degree
☐ Bachelor's Degree
☐ Master's Degree
☐ Doctorate Degree

Number of Years in Respiratory Care

☐ 0-2 years
☐ 3-5 years
☐ 6-10 years
☐ 11-15 Years
☐ 16 years or more

Job Status

☐ Full Time
☐ Part Time

Credentials

☐ RRT
☐ CRTT
☐ Physician
☐ CRNA
☐ RN
☐ LVN/LPN
☐ CPFT
☐ RPFT
☐ Perinatal/Pediatric

Salary

☐ Less than $10,000
☐ $10,001-$20,000
☐ $20,001-$30,000
☐ $30,001-$40,000
☐ $40,000 or more

PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE journal and *AARC Times* magazine includes an allocation of $11.50 from my dues for each of these publications.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%.

Signature ______________________________

Date ______________________________

Membership Fees

Payment must accompany your application to the AARC. Fees are for 12 months. (NOTE: Renewal fees are $75.00 Active, Associate-Industrial or Associate-Physician, or Special status; $90.00 for Associate-Foreign status; and $45.00 for Student status).

☐ Active	$ 87.50
☐ Associate (Industrial or Physician)	$ 87.50
☐ Associate (Foreign)	$102.50
☐ Special	$ 87.50
☐ Student	$ 45.00
TOTAL	$__________

Specialty Sections

Established to recognize the specialty areas of respiratory care, these sections publish a bi-monthly newsletter that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

☐ Adult Acute Care Section	$15.00
☐ Education Section	$20.00
☐ Perinatal-Pediatric Section	$15.00
☐ Diagnostics Section	$15.00
☐ Continuing Care-Rehabilitation Section	$15.00
☐ Management Section	$20.00
☐ Transport Section	$15.00
☐ Home Care Section	$15.00
☐ Subacute Care Section	$15.00
TOTAL	$__________

GRAND TOTAL = Membership Fee plus optional sections $__________

☐ Total Amount Enclosed/Charged $__________________
☐ Please charge my dues (see below)

To charge your dues, complete the following:
☐ MasterCard
☐ Visa

AARC

Card Number

__ __ __ __ __ __ __ __ __ __ __ __ __ __ __ __

Card Expires _______/______

Signature ______________________________________

Mail application and appropriate fees to:
American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 484-2720

MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting
by health professionals of adverse
events and product problems

Page ____ of ____

Form Approved: OMB No. 0910-0291 Expires: 4/30/96
See OMB statement on reverse

FDA Use Only (Resp Care)

Triage unit
sequence #

A. Patient information

1. **Patient identifier**
In confidence

2. **Age at time of event:**
or
Date of birth:

3. **Sex**
☐ female
☐ male

4. **Weight**
____ lbs
or
____ kgs

B. Adverse event or product problem

1. ☐ **Adverse event** and/or ☐ **Product problem** (e.g., defects/malfunctions)

2. **Outcomes attributed to adverse event**
(check all that apply)
☐ death ____ (mo/day/yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other: ____

3. **Date of event** (mo/day/yr)

4. **Date of this report** (mo/day/yr)

5. **Describe event or problem**

6. **Relevant tests/laboratory data**, including dates

7. **Other relevant history, including preexisting medical conditions** (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. **Name** (give labeled strength & mfr/labeler, if known)
#1
#2

2. **Dose, frequency & route used**
#1
#2

3. **Therapy dates** (if unknown, give duration)
from/to (or best estimate)
#1
#2

4. **Diagnosis for use** (indication)
#1
#2

5. **Event abated after use stopped or dose reduced**
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. **Lot #** (if known)
#1
#2

7. **Exp. date** (if known)
#1
#2

8. **Event reappeared after reintroduction**
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9. **NDC #** (for product problems only)
– –

10. **Concomitant medical products** and therapy dates (exclude treatment of event)

D. Suspect medical device

1. **Brand name**

2. **Type of device**

3. **Manufacturer name & address**

4. **Operator of device**
☐ health professional
☐ lay user/patient
☐ other: ____

5. **Expiration date** (mo/day/yr)

6.
model # ____
catalog # ____
serial # ____
lot # ____
other #

7. **If implanted, give date** (mo/day/yr)

8. **If explanted, give date** (mo/day/yr)

9. **Device available for evaluation?** (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ____ (mo/day/yr)

10. **Concomitant medical products** and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. **Name & address**

phone #

2. **Health professional?**
☐ yes ☐ no

3. **Occupation**

4. **Also reported to**
☐ manufacturer
☐ user facility
☐ distributor

5. **If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box.** ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

DA Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

• 1-800-FDA-0178	to FAX report
• 1-800-FDA-7737	to report by modem
• 1-800-FDA-1088	to report by phone or for more information
• 1-800-822-7967	for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to:

DHHS Reports Clearance Office
Paperwork Reduction Project (0910-0291)
Hubert H Humphrey Building, Room 531-H
200 Independence Avenue, S W
Washington, DC 20201

'An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number.'

Please do NOT return this form to either of these addresses.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service • Food and Drug Administration

FDA Form 3500-back

Please Use Address Provided Below – Just Fold In Thirds, Tape and Mail

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Rockville, MD 20857

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MEDWATCH
The FDA Medical Products Reporting Program
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20852-9787

call for abstracts

1998 RESPIRATORY CARE OPEN FORUM

The American Association for Respiratory Care and its science journal, Respiratory Care, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the Open Forum during the AARC International Respiratory Congress in Atlanta, Georgia, November 7–10, 1998. Accepted abstracts will be published in the October 1998 issue of Respiratory Care. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract is the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the Open Forum. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write general statements, such as "Results will be presented" or "Significance will be discussed."

ESSENTIAL CONTENT ELEMENTS

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocol evaluation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: relevant basic information important to understanding the case. (2) Case Summary: patient data and response, details of interventions. (3) Discussion: content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

FORMAT AND TYPING INSTRUCTIONS

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be *at least* 10 points. *Abstracts should be 400 words or less and may have 1 clear, concise table or figure.* A font like Helvetica or Geneva makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter's name. Type or electronically print the abstract *single spaced in a single paragraph in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. *Data may be submitted in table form, or a simple figure may be included provided it fits within the space allotted. No figure, illustration, or table is to be attached to the abstract form.* Provide all author information requested. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of Respiratory Care at (972) 406-4667.

Early Deadline Allowing Revision. Authors may choose to submit abstracts early. Abstracts postmarked by February 16, 1998 will be reviewed and the authors notified by letter only to be mailed by March 20, 1998. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the Final Deadline (May 4, 1998).

Final Deadline. The mandatory Final Deadline is May 4, 1998 (postmark). Authors will be notified of acceptance or rejection *by letter only.* These letters will be mailed by July 20, 1998.

Mailing Instructions. Mail (do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

1998 Respiratory Care Open Forum
11030 Ables Lane
Dallas TX 75229-4593

submit your OPEN FORUM abstract electronically
visit www.rcjournal.com

1998 RESPIRATORY CARE OPEN FORUM Abstract Form

Horizontal line length: 13 cm or 5.125"

18.8 cm or 7.4"

13.9 cm or 5.5"

1. Title must be in all upper case (capital) letters, authors' full names and text in upper and lower case.
2. Follow title with all authors' names, including credentials (underline presenter's name), institution, and location.
3. Do not justify (ie, leave a 'ragged' right margin).
4. **Do not use type size less than 10 points.** (Do not exceed 400 words.)
5. All text and the table, or figure, must fit into the rectangle shown. (Use only 1 clear, concise table or figure.)
6. Submit 2 clean copies. This form may be photocopied if multiple abstracts are to be submitted.

Mail original & 1 photocopy (along with postage-paid postcard) to

1988 RESPIRATORY CARE OPEN FORUM
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RESPIRATORY CARE

Manuscript Preparation Guide

General Information

RESPIRATORY CARE welcomes original manuscripts related to the science and technology of respiratory care and prepared according to these Instructions and the *Uniform Requirements for Manuscripts Submitted to Biomedical Journals* [Respir Care 1997; 42(6):623-634]. Manuscripts are blinded and reviewed by professionals who are experts in their fields. Authors are responsible for all aspects of the manuscript and receive galleys to proofread before publication. Each accepted manuscript is copyedited so that its message is clear and it conforms to the Journal's style. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission.

Editorial consultation is available at any stage of planning or writing. On request, specific guidance is provided for all publication categories. To receive these Instructions and related materials, write to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104, call (206) 223-0558, or fax (206) 223-0563.

Publication Categories & Structure

Research Article: A report of an original investigation (a study). It includes a Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Evaluation of Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification. It has a Title Page, Abstract, Introduction, Description of Device/Method/Technique, Evaluation Methods, Evaluation Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions. Comparative cost data should be included wherever possible.

Case Report: A report of a clinical case that is uncommon, or was managed in a new way, or is exceptionally instructive. All authors must be associated with the case. A case-managing physician must either be an author or furnish a letter approving the manuscript. Its components are Title Page, Abstract, Introduction, Case Summary, Discussion, References, Tables, Figures, and Figure Captions.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles. Title Page, Outline, Introduction, Review of the Literature, Summary, Acknowledgments, References. Tables, Appendices, and Figures and Captions may be included.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles.

Update: A report of subsequent developments in a topic that has been critically reviewed in this Journal or elsewhere.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Title Page, Text, References, Tables, and Illustrations may be included.

Special Article: A pertinent paper not fitting one of the foregoing categories may be acceptable as a Special Article. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper drawing attention to a pertinent concern; it may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A signed communication, marked "For publication," about prior publications in this Journal or about other pertinent topics. Tables and illustrations may be included.

Blood Gas Corner: A brief, instructive case report involving blood gas values—with Questions, Answers, and Discussion.

Drug Capsule: A mini-review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, and pharmacotherapy.

Graphics Corner: A brief case report incorporating waveforms for monitoring or diagnosis—with Questions, Answers, and Discussion.

Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Cardiorespiratory Interactions. A case report demonstrating the interaction between the cardiovascular and respiratory systems. It should be a patient-care scenario; however, the case—the central theme—is the systems interaction. CRI is characterized by figures, equations, and a glossary. See the March 1996 Issue of RESPIRATORY CARE for more detail.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs; may involve imaging techniques other than conventional chest radiography.

Review of Book, Film, Tape, or Software: A balanced, critical review of a recent release.

Preparing the Manuscript

Print on one side of white bond paper, 8.5 in. x 11 in. (216 x 279 mm) with margins of at least 1 in. (25 mm) on all sides of the page. Use ***double-spacing*** throughout the ***entire*** manuscript. Use a standard font (eg, Times, Helvetica, or Courier) at least 10 points in size, and do not use italics except for special emphasis. Number all pages in upper-right corners. Indent paragraphs 5 spaces. Do ***not justify***. **Do *not* put authors' names, institutional affiliations or allusions to institutional affiliations in the text, or other identification anywhere except on the title page.** Repeat title only (no authors) on the abstract page. Begin each of the following on a new page: Title Page, Abstract, Text, Product Sources List, Acknowledgments, References, each Table, and each Appendix. Use standard English in the first person and active voice.

Center main section headings on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin subheadings at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis).

References. Cite only published works as references. Manuscripts accepted but not yet published may be cited as references: designate the accepting journal, followed by (in press), and provide 3 copies of the in-press article for reviewer inspection. Cite references in the text with superscript numerals. Assign numbers in the order that references are first cited. On the reference page, list the cited works in numerical order. Follow the Journal's style for references. Abbreviate journal names as in *Index Medicus*. List all authors.

Article in a journal carrying pagination throughout volume:

> Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

> Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

> American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

> Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986; 89(3 Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

> Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

> Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

> Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

> Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

> Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

> DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura; 1990. p. 76-85.

Corporate author book:

> American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

Chapter in book with editor(s):

> Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott; 1977:26-42.

Tables. Use consecutively numbered tables to display information. Start each table on a separate page. Number and title the table and give each column a brief heading. Place explanations in footnotes, including all nonstandard abbreviations and symbols. Key the footnotes with conventional designations (*, †, ‡, §, ||, ¶, **, ††) in consistent order, placing them superscript in the table body. Do not use horizontal or vertical rules or borders. Do not submit tables as photographs, reduced in size, or on oversize paper. Use the same typeface as in the text.

Illustrations. Graphs, line drawings, photographs, and radiographs are figures. Use only illustrations that clarify and augment the text. Number them consecutively as Fig. 1, Fig. 2, and so forth according to the order by which they are mentioned in the text. Be sure all figures are cited. If any figure was previously published, include copyright holder's written permission to reproduce. Figures for publication must be of professional quality. Data for the original graphs should be available to the Editor upon request. If color is essential, consult the Editor for more information. In reports of animal experiments, use schematic drawings, not photographs. A letter of consent must accompany any photograph of a person. Do not place titles and detailed explanations on figures; put this information in figure captions. If possible, submit radiographs as prints and full-size copies of film.

Drugs. Identify precisely all drugs and chemicals used, giving generic names, doses, and routes of administration. If desired, brand names may be given in parentheses after generic names. Drugs should be listed on the product-sources page.

Commercial Products. In parentheses in the text, identify any commercial product (including model number if applicable) the first time it is mentioned, giving the manufacturer's name, city, and state or country. If four or more products are mentioned, do not list any manufacturers in the text; instead, list them on a Product Sources page at the end of the text, before the References. Provide model numbers when available and manufacturer's suggested price, if the study has cost implications.

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AARC & AFFILIATES

April 3: Pineywoods District of the Texas Society
10th annual seminar, A Practical Approach to Respiratory Care, Pitser Garrison Civic Center, Lufkin, Texas.
Contact: (409) 639-7006

April 15–16: Utah Society
1998 Annual Convention & Exhibition, Pulmonopoly—Insuring Your Investment in Health Care Futures, Salt Palace, Salt Lake City, Utah.
Contact: Kirt Day—(801) 398-2956, fax: (801) 629-5487; e-mail: mkkday@ihc.com; web: **www.med.utah.edu/usrc**

April 15–17: Kansas Society
21st Annual Educational Seminar—From Silver to Gold, Lawrence, Kansas.
CRCE: Credit has been requested.
Contact: Pat Munzer—(785) 231-1010, ext 1404; e-mail: zzmunz@washburn.edu

April 21–24: Michigan Society
Anual scientific symposium, Illuminating Your Professional Horizons, Holiday Inn, Lansing, Michigan.
Contact: (517) 336-7605

April 22–23: Pennsylvania Society
1st annual Eastern Regional Conference & Exhibition—Pride & Professionalism, Holiday Inn, King of Prussia, Pennsylvania.
Contact: Angie Herstine—(609) 784-0340 or Cathy Hurley—(215) 453-4185

April 22–24: Iowa Society
20th Annual Iowa Lung Conference: Evolutions of Respiratory Care—Past, Present, & Future, Embassy Suites on the River, Des Moines, Iowa.
Contact: (515) 247-4050

May 3–5: Washington Society
25th Annual Pacific Northwest Regional Respiratory Care Conference, Cavanaughs Inn at the Park, Spokane, Washington. Neil R MacIntyre MD will be the featured speaker.
CRCE: Credit available.
Contact: Jan Tucker—(800) 464-2126

May 5–6: Connecticut Society
Annual Super Symposium, Foxwood Resort and Casino, Ledyard, Connecticut.
Contact: Karen Milikowski—(860) 647-6193, fax (860) 647-6370; e-mail: ma_milikowsk@commnet.edu

May 6–8: South Dakota Society
Annual meeting, Howard Johnson Hotel, Sioux Falls, South Dakota. Neil R MacIntyre MD will be the featured speaker.
Contact: Gordy Gunderson—(605) 333-5207 or Ruth Krueger—(605) 333-6514

May 6–8: Tennessee Society
Annual Convention & Exhibition, Navigating Uncharted Waters, MeadowView Resort & Convention Center, Kingsport, Tennessee.
CRCE: 13 contact hours are available.
Contact: Colleen Schabacker—(615) 384-1731

May 12–15: Region II—Indiana, Kentucky, & Ohio Societies
25th Annual Region II Conference, Hyatt Regency Hotel & the Albert B Sabin Convention Center, Cincinnati, Ohio.
Contact: (800) 691-3046, Mail Box #3, or **www.bright.net/~dsibb/reg2rc.htm**

May 20–22: Southwest Region of the Texas Society
27th Annual Respiratory Care Symposium & Technical Exhibition, El Paso Community College's Transmountain Campus, El Paso, Texas.
CRCE: 12-16 hours are available.
Contact: Yvonne Banegas—(915) 577-6563

May 22: Alamo District of the Texas Society, The University of Texas Health Science Center at San Antonio, & Wilford Hall Medical Center
3rd Annual Riverwalk Respiratory Care Symposium, San Antonio Marriott Riverwalk Hotel, San Antonio, Texas.
CRCE: 6 credits available.
Contact: (210) 567-3706

June 25–27: California Society
30th Annual Convention: Thirty Something: Three Decades of Progress & Change, Monterey Marriott Hotel, Monterey, California.
CRCE: 17 hours.
Contact: (805) 723-1180

OTHER MEETINGS

April 9–10: InfoCare
Annual Adult Respiratory/Critical Care Symposium, Sheraton Fiesta Beach Resort, South Padre Island, Texas.
Contact: InfoCare—(956) 233-5645; fax: (956) 233-1010.

April 15–17: National Subacute Care Association & Contemporary Long-Term Care
Subacute Care '98—Paths to the Future, Hyatt Regency, Chicago, Illinois.
Contact: (800) 765-7616.

April 22–24: Ohio Sleep Medicine Institute
Center of Sleep Medicine Excellence™—TechShop '98—3-day, introductory-level, training for respiratory therapists & sleep technologists seeking to enhance their skills in sleep medicine, Columbus, Ohio.
Contact: Crystal Moore—(614) 766-0773; e-mail: sleepohio@aol.com.

April 24–29: American Lung Association & the American Thoracic Society
1998 ALA/ATS International Conference, Chicago, Illinois.
Contact: (212) 315-6440.

April 30–May 1: American Lung Association of Nevada
13th Annual Respiratory Health Conference, Tropicana Resort & Casino, Las Vegas, Nevada.
Contact: (702) 431-6333

May 15–16: American Society for Testing Materials & EMS Magazine Exposition
The ASTM Committee F-30 on Emergency Medical Services, Nashville Convention Center, Nashville, Tennessee.
Contact: Len Morrissey—(610) 832-9730; e-mail: lmorriss@ astm.org.

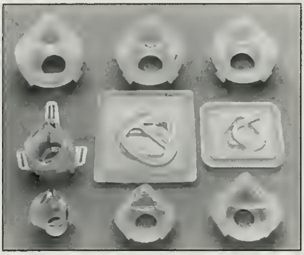
May 21: Respiratory Care Services of Arkansas Children's Hospital
Diamond Conference—Annual Pediatric-Neonatal Cardiopulmonary Critical Care Conference, Little Rock, Arkansas. Keynote speaker Forrest M Bird MD PhD & a special session for nurses.
CRCE: Category I credits, contact hours for nurses, and physician CMEs are available.
Contact: Rachel Bull—(501) 320-3530; fax: (501) 320-1887; e-mail: manders@exchange.ach.uams.edu; web: **www.ach.uams.edu/announce/diamond/**

August 7–9: American Lung Association of Southeast Florida, the American Heart Association, & the Florida Association of Cardiovascular and Pulmonary Rehabilitation
4thannual Cardiopulmonary Rehabilitation Conference, Jupiter Beach Resort, Jupiter, Florida.
CRCE: Credits are available.
Contact: Carol Boyd—(561) 659-7644 by July 31

September 18–20: ELSO
1998 Annual ELSO Meeting, Placido del Rio Hilton, San Antonio, Texas.
Contact: Phoebe Hankins—(734) 998-6600; fax: (734) 998-6602; e-mail: phankins@umich.edu; web: **www.med.umich. edu/elso**

New Products & Services

News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE, New Products & Services Dept, 11030 Ables Lane, Dallas TX 75229-4593. The Reader Service Card immediately follows the authors & advertisers index at the back of the Journal.



CPAP MASK SEAL. Hans Rudolph Inc offers the Ultimate Seal™ line of masks. The molded hydrogel masks may provide greater patient comfort, allow the facial skin to breathe, and form a leak-free barrier between the patient's face and the sealing surface of the mask. According to Hans Rudolph, the moisture-permeable gel technology also promotes wound healing and wound prevention. Ultimate Seal products are configured to match the geometry of standard-type and AirSep Corporation nasal CPAP masks and the Rudolph full-face masks. Circle Reader Service Number 160 for further details, or send your request electronically via "Advertisers Online" at www.aarc.org/resources.html.

TELECOMMUNICATION SYSTEM. Nellcor Puritan Bennett announces the Telesurveillance® telecommunication system. Using an existing telephone line in the patient's home and a dedicated phone line at the provider location, the system is capable of 2-way communication between the patient and the home care provider. In addition, Telesurveillance offers real-time verification of device status and patient oxygen therapy compliance. For more information, circle number 161 on the Reader Service Card in this issue, or send your request electronically using the "Advertisers Online" section at www.aarc.org/resources.html.

BLOOD ANALYZER. Radiometer America Inc introduces the ABL™555 pH/Blood Gas, Electrolyte and Metabolite Analyzer. According to company literature, the ABL555 offers up to 30 measured parameter configurations for STAT whole blood testing and offers exclusive features such as interference-free accuracy, multiprofile testing option with reduced sample volumes, and flexible upgrade options for the ABL505 Analyzer. Radiometer says the parameter combination options include pH, P_{CO_2}, P_{O_2}, hematocrit, Na^+, K^+, Ca^{++} or Cl^- or glucose or lactate. For additional details, circle number 162 on the Reader Service Card in this issue, or send your request electronically via "Advertisers Online" at www.aarc.org/resources.html.

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP) SYSTEM. Respironics releases the Virtuoso® LX Smart CPAP System. The device is an automatically adjusting CPAP system for the treatment of adult obstructive sleep apnea. In a press release from Respironics, the system is reported to be quiet, compact, lightweight (3.5 lbs), portable, and able to operate on 115 and 230 volts alternating current or 12 volts direct current power. The patented Virtuoso LX System offers a range of pressures, from 3 to 20 cm H_2O, and functions in 3 modes of therapy: CPAP, auto-CPAP, and split night with auto-CPAP. For more information from Respironics, circle Reader Service Number 163, or send your request electronically using the "Advertisers Online" section at www.aarc.org/resources. html.

LIQUID AEROSOL. AeroGen has developed a liquid aerosol technology for 2 inhalation drug delivery systems: AeroNeb,™ a continuous delivery compact nebulizer, and AeroDose,™ a multidose, breath-actuated inhaler designed for use with a variety of drugs. AeroGen reports that they have received Food & Drug Administration approval for the AeroNeb,™ and will promote the nebulizer as a cost-effective alternative for the U.S. home care market. The low velocity, propellant-free AeroDose™ inhaler can be configured for use as a single-dose unit or with a multidose canister. For details from AeroGen, circle number 164 on the Reader Service Card in this issue, or send your request electronically via "Advertisers Online" at www.aarc.org/resources.html.

FENOFIBRATE CAPSULES. Abbott Laboratories announces Food & Drug Administration marketing approval for Tricor,™ a micronized form of the drug fenofibrate for patients with high triglyceride levels. Tricor™ may be indicated for the treatment of adults who are at risk for pancreatitis with Types IV and V hyperlipidemia that is not appropriately controlled by diet alone. For more information from Abbott about indications, contraindications, and side effects, circle Reader Service Number 165, or send your request electronically via "Advertisers Online" at www.aarc.org/resources.html.

INHALATION SOLUTION. Roxane Laboratories has been given Food & Drug Administration clearance to relaunch and market Isoetharine Inhalation Solution USP, 1% (previously marketed as Bronkosol® by Sanofi Winthrop). According to Roxane, the product will be available in 10-mL and 30-mL multidose bottles with calibrated droppers. The company says it is committed to long-term manufacturing of the raw material as well as supplying the actual product. Indications, contraindications, and side effects informaion is available from Roxane Laboratories. Circle 166 on the Reader Service Card in this issue, or send your request electronically by way of "Advertisers Online" at www.aarc.org/resources.html.

DETACHABLE FACE MASKS. HealthScan Products Inc introduces detachable face masks for use with OptiChamber® Valved Holding Chamber. The face masks, according to HealthScan, are helpful in administering inhaler medication to infants, toddlers, and other patients who have difficulty using the mouthpiece on a holding chamber, such as the OptiChamber. The masks, available for different facial shapes and sizes, provide a seal that may prevent medication leaks. To find out more from HealthScan, Circle Reader Service Number 167, or request information electronically via "Advertisers Online" at www.aarc.org/resources.html.

Policy and Procedure Manual

Save time and money by making your department more efficient with the *Policy and Procedure Manual.* Its 130 pages of policies and procedures cover the aspects of administrative and clinical respiratory care for both adult and pediatric practice. Sections on administrative policies, therapeutics, clinical monitoring, and mechanical ventilation. Standardized formats include objectives, indications, equipment, policies, contraindications, troubleshooting, procedures, hazards, and assessment of effectiveness.

Item BK6 $60 ($70 nonmembers)

Uniform Reporting Manual

The *Uniform Reporting Manual* provides you with nationally recognized standards for documenting workload units and time standards. Includes patient assessment activities and covers bronchial hygiene, supplemental oxygen, airway care, diagnostic tests, and cardiovascular diagnostics. In addition, there are chapters on "Clinical Activities Without Time Standard" and "Management Support Activities." *By the AARC. 1990, updated 1993. Binder, 165 pages.*

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A Study of Chronic Ventilator Patients in the Hospital

Chronic ventilator-dependent patients are costing American hospitals more than $9 million per day according to this Gallup study conducted for the AARC. This important study provides information on patients who depend on life-support systems; why, how, and where they are being treated; and the cost of treatment. *47 pages, 9 tables, 12 figures.*

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Item BK12 $35 ($50 nonmembers)

A Study of Respiratory Care Practice

This study examines the practice of respiratory care in today's health care environment and how hospital reorganization is affecting the profession. Includes chapters on medical direction, current respiratory care services, nontraditional services, respiratory care protocols, and hospital service reorganization. *38 pages, 34 tables, 15 charts.*

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www.sleepapnea.org

American Sleep Disorders Association
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www.asda.org; e-mail: asda@asda.org

National Center on Sleep Disorders Research
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www.nhlbi.nih.gov/nhlbi/sleep/sleep.htm

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New *Federal Register* Notices Now Available

The Center for Devices and Radiological Health announces the publication of new Facts-on-Demand FOD notices in the *Federal Register*. The new publications: FOD#774—Medical Devices; Preemption of State Product Liability Claims; Proposed Rule; FOD#607—Rebuilders, Reconditioners, Services, and "As Is" Remarketers of Medical Devices; Review and Revision of Compliance Policy Guides and Regulatory Requirements; Request for Comments and Information; Proposed Rule; and FOD#513—Medical Devices; Reports of Corrections and Removals; Stay of Effective Date of Information Collection Requirements; Stay of Effective Date of Final Regulation. For more information about Facts-on-Demand call (800) 899-0381 or (301) 826-0111. The FOD system is also on the Internet at www.fda.gov/cdrh/fedregin.html.

NAMES 1998 Education, Conference Schedule Set

The National Association for Medical Equipment Services (NAMES) announces its 1998 national conferences and regional education seminars. For information about upcoming events, call the NAMES Education & Meeting Department at (703) 836-6263, or visit the web site: www.names.org.

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	November 14, 1998 Application Deadline: September 1, 1998	100 (new applicant)* 60 (reapplicant)*
RRT Examination	June 6, 1998 Application Deadline: February 1, 1998	120 Written only (new applicant) 80 Written only (reapplicant)
		130 CSE only (all applicants)
	December 5, 1998 Application Deadline: August 1, 1998	250 Both (new applicant) 210 Both (reapplicant)
CPFT Examination	June 6, 1998 Application Deadline: April 1, 1998	130 (new applicant) 100 (reapplicant)
RPFT Examination	December 5, 1998 Application Deadline: September 1, 1998	180 (new applicant) 150 (reapplicant)
Perinatal/Pediatric Respiratory Care Specialty Examination	March 14, 1998 Application Deadline: November 1, 1998	180 (new applicant) 150 (reapplicant)

*In 1999, this examination fee will increase by $20.

For information about other services or fees, write to the National Board for Respiratory Care,
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Authors in This Issue

Atwood Jr, Charles W 307
Chediak, Alejandro D 265
Minkley, Pamela 296
Phillips, Barbara A 288
Stoller, James K 264
Strohl, Kingman P 264, 277
Strollo Jr, Patrick J 264
Studnicki, Karen A 318

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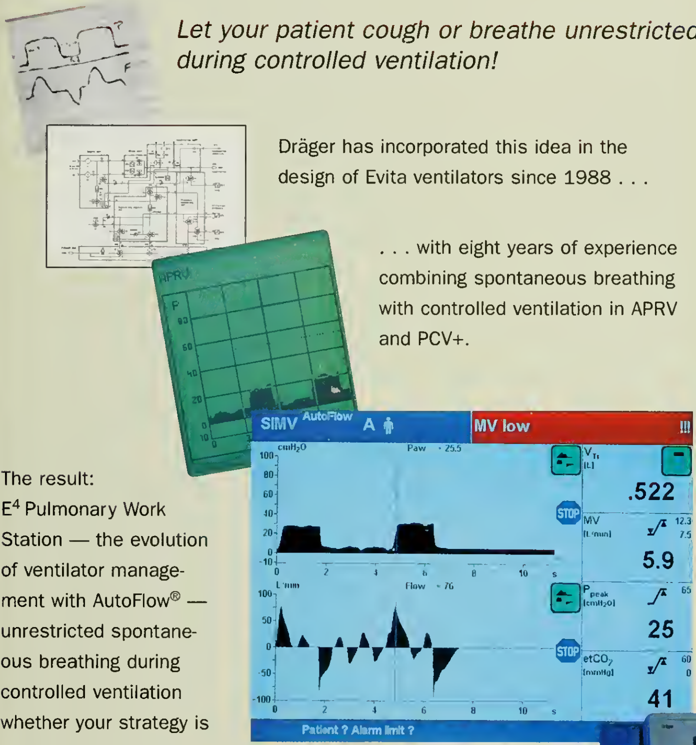

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